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AMERICAN JOURNAL OF OPHTHALMOLOGY

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DEPARTMENTS

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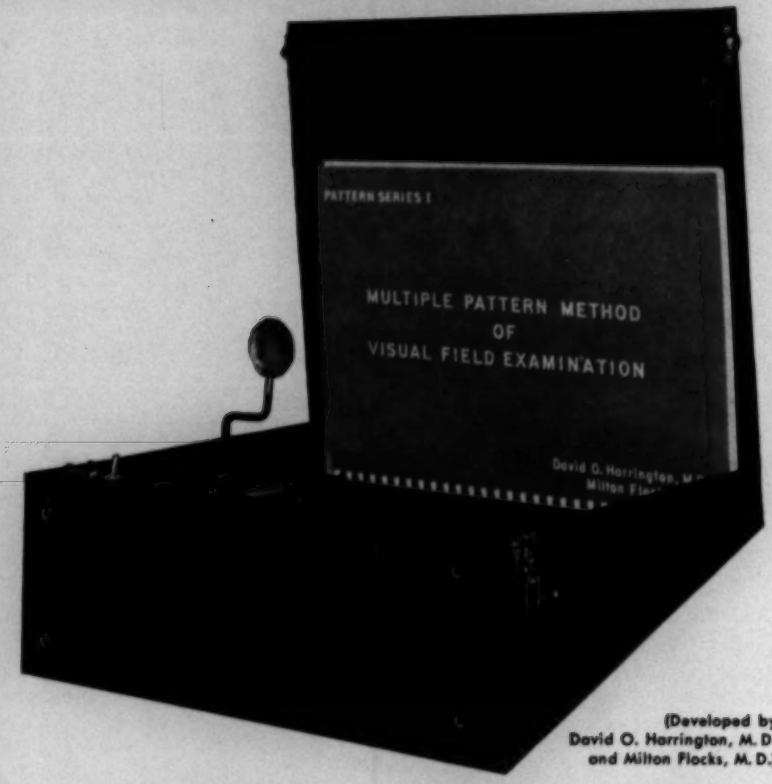

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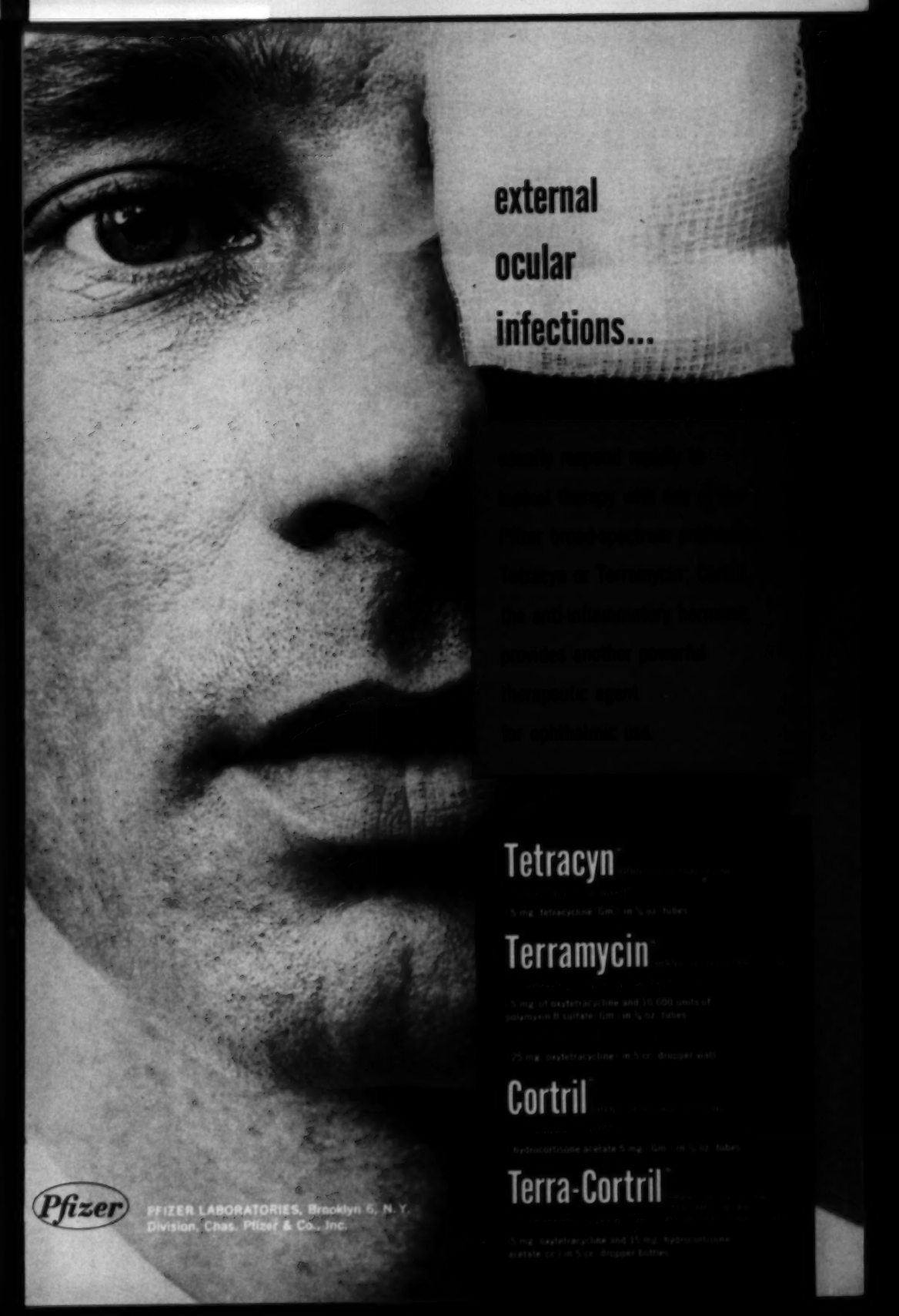
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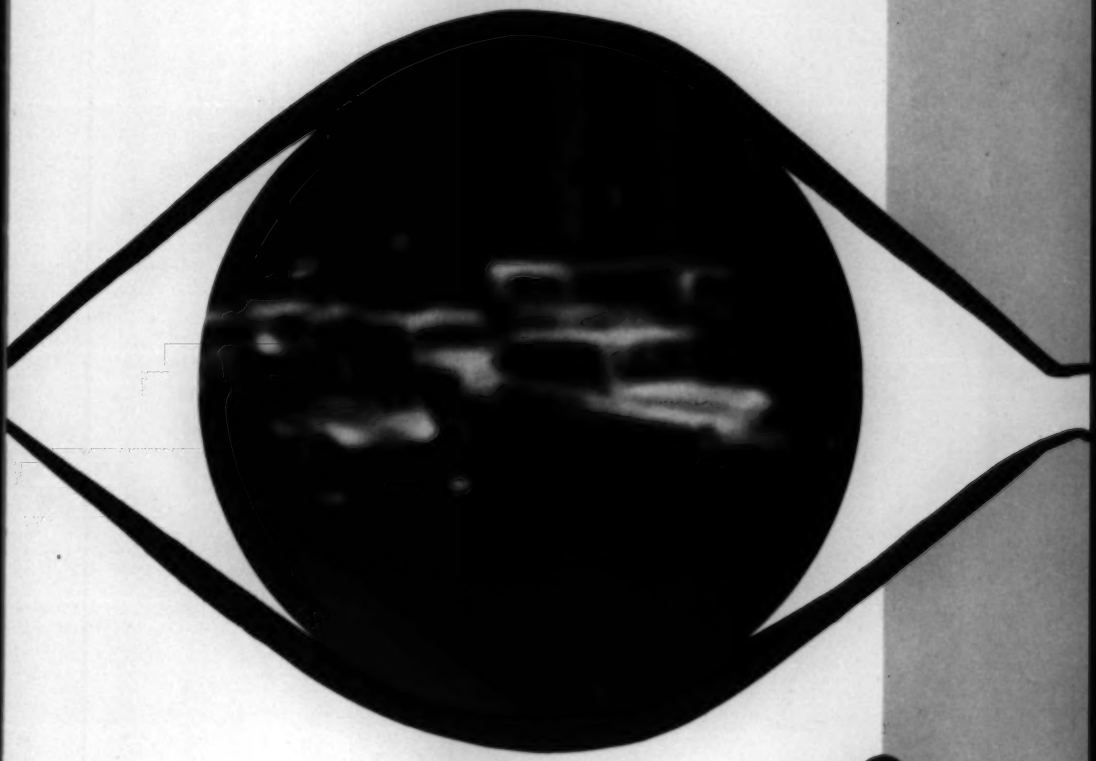

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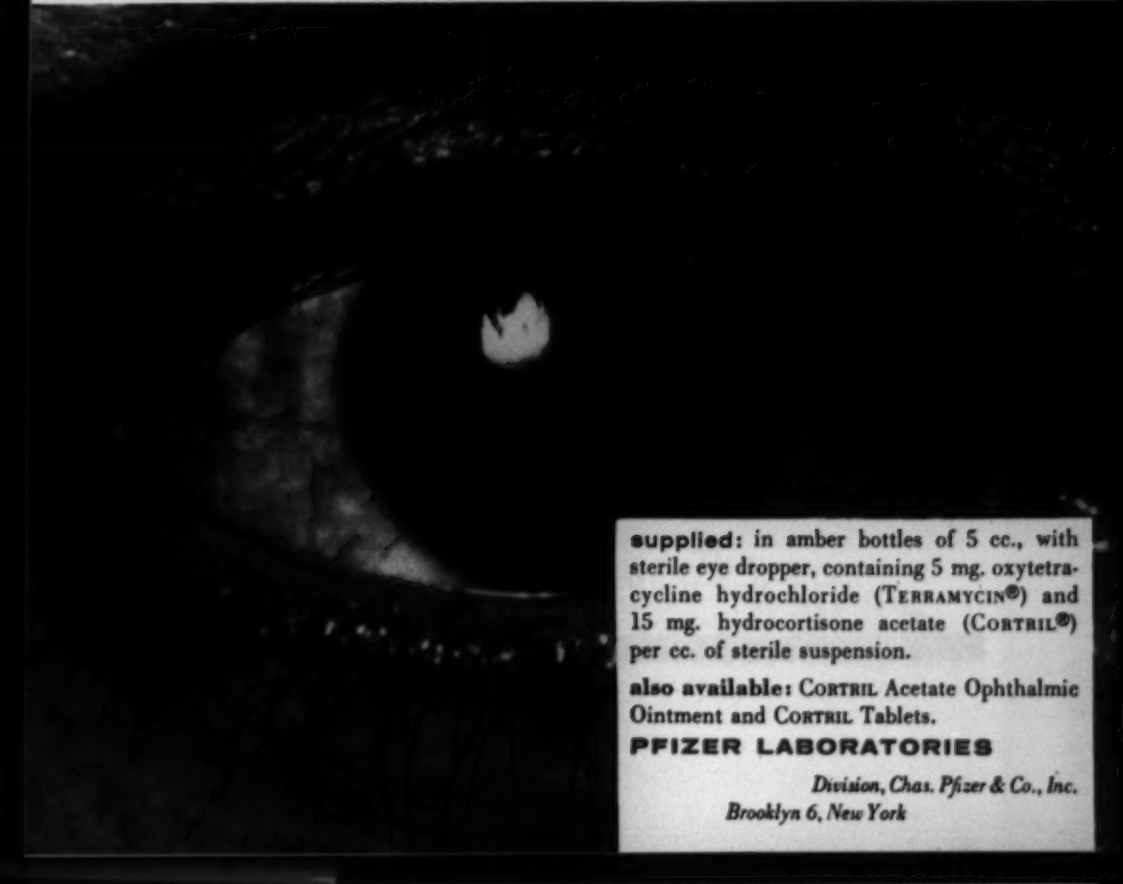

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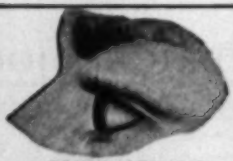

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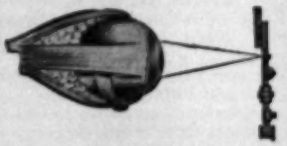

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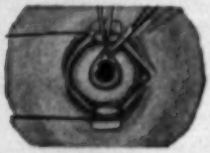

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TONOGRAPHY IN SOME PROVOCATIVE TESTS FOR GLAUCOMA*

HELVI SWANLJUNG, M.D., AND FREDERICK C. BLODI, M.D.
Iowa City, Iowa

Any method of examination that will enable us to make an earlier diagnosis of glaucoma warrants further investigation and trial. A great number of provocation tests have been employed for this purpose with varying results. Frequent overenthusiasm has been followed by disappointment and many of these tests have fallen into disrepute. However, we believe that a number of provocative tests can be judiciously used with advantage and that most of the misunderstanding surrounding them can be cleared by a better understanding of some of their basic properties.

We are interested in two main properties of any provocative test. One is its reliability, the other one is the mechanism by which it increases the intraocular pressure. The reliability of a provocative test is the relative number of positive results in a series of early, untreated cases of glaucoma. Unfortunately, there is no single test with a reliability of 100 percent. This is perhaps due to the fact that the increased intraocular pressure is not always due to the same pathologic process. The degree of reliability can be estimated only after examining a large series of cases. This has been done by pooling patients from various clinics.[1,2] The degree of reliability varies with the test. It is only five percent for the caffeine test and about 15 percent for the lability test. That means only five or 15 patients out of 100 early glaucomas will give a significant rise of the intraocular pressure after caffeine or with the lability test. This percentage is obviously too low to be of any practical value and we will usually resort to provocative tests of a higher degree of reliability.

The mechanism of some provocative tests has been studied extensively. In the water-drinking test for instance the increase in the intraocular pressure can be correlated to the fall of the osmotic pressure and the sodium in the blood.[3,4] In the lability test we have partly an embarrassment of venous outflow from the head. The variations in the effective mechanism of these tests account for the incomplete parallelism among them. One eye may give a positive result with the water-drinking test, but an insignificant increase in intraocular pressure after caffeine and vice versa. This would make it advisable to use the whole battery of provocative tests in a dubious case of glaucoma, provided one test after the other gave a negative result. One positive test would obviously make any further testing unnecessary. In any case, this form of diagnosis would be highly impractical and we have to select the tests which are most practical, have a high degree of reliability, and have an effective mechanism which would lead us to expect an increased intraocular pressure in a particular type of glaucoma. It is certainly unlikely for example, that an eye with chronic simple glaucoma would give a positive result to a dark-room test.

We have selected for our study two provocative tests of high reliability in eyes with

* From the Department of Ophthalmology, College of Medicine, State University of Iowa. Presented at the meeting of the Midwestern Section, Association for Research in Ophthalmology.

chronic simple glaucoma. The first test is the water-drinking test. This test has been extensively studied. It is effective because of a lowering of the osmotic pressure in the plasma which increases the relative hypertonicity of the aqueous. This test has many advantages. It is easy to administer and entails no discomfort for the patient. Its reliability has been estimated from 27 to 67 percent.[5]

Our plan was to do tonography before and during the test. This has already been done by A. DeRoetth, Jr., who found an increased rate of flow of aqueous in glaucomatous and normal eyes.[6,7] The facility of aqueous outflow remained unchanged in glaucomatous eyes but increased in normal eyes. It was our plan to check these findings and in addition to evaluate the administration of Diamox during the water-drinking test.* If the increased intraocular pressure during a positive water-drinking test is indeed caused by an uncompensated increased rate of aqueous flow it should be possible to suppress this reaction completely or partially by the simultaneous administration of this potent carbonic-anhydrase inhibitor.

The other provocative test studied was the Priscoline test. This test was described by Leydhecker in 1954.[8] It consists of a subconjunctival injection of the vasodilator. A normal eye will be able to compensate the sudden hyperemia, a glaucomatous eye will experience a marked rise in pressure. The advantages of this test are manifold. This is a provocative test which acts on the eye directly and not via systemic effects of blood pressure, blood flow, osmotic pressure, and so forth. These systemic effects may be influenced by many factors, such as the condition of the kidney, the heart, and so on. The Priscoline test is relatively independent from these factors. Another great advantage is the fact that one eye alone can be tested while the other eye remains unaffected. The other eye may serve as a control or may be entirely unsuited for a provocative test. So far, no tonographic studies on this test have been reported.

* This procedure was suggested by Dr. Bernard Schwartz, Iowa City, Iowa.

Method

The patients for this study were taken from the eye clinic of the University Hospital and the eye service of the Veterans Administration Hospital in Iowa City. The patients had a diagnosed or suspected chronic simple (open-angle) glaucoma. Evaluated were only eyes without previous antiglaucomatous surgery. Miotics were discontinued at least 24 hours prior to the provocative tests. Only two of the 37 patients were entirely control cases without any symptoms or signs of glaucoma.

The intraocular pressure was measured with the Schiøtz tonometer, the patient lying flat on his bed. The scale readings were transposed into or recorded in millimeters of mercury according to the new scale published by the American Academy of Ophthalmology and Otoloryngology.

The tonography was performed according to the method of Grant. Prior to the control tonography two tonometric readings were taken in 20- to 30-minute intervals in order to detect any pronounced spontaneous variation. An interval of five minutes was always allowed between the tonography of the right and the left eye. This was done to eliminate the tonography of one eye influencing the aqueous flow in the other eye. The right eye was always tested first, except after an injection of Priscoline where the injected eye was tested first. However, this interval of five minutes is probably not enough, as it was found that the intraocular pressure in the left eye was consistently lower than in the right. This does not mean that the resistance to outflow and the aqueous flow were necessarily also influenced.

The water-drinking test was done at approximately the same time of the day as the preceding control tonography. In a few cases it was impossible to keep the patient overnight. There, the water-drinking test was done one hour after the control tonography.

The patient was asked to drink 1,000 cc. water within two to four minutes. This was done on an empty stomach. Tonography was done either 20 and 60 minutes later or only once, 30 minutes later. It was felt that the two tonographies did not differ appreciably. Therefore, only one tonography was done after the drinking of water in the majority of cases.

For the Priscoline test, one cc. of the solution containing 0.01 gm. of the active drug in distilled water was injected subconjunctivally about 10 mm. away from the limbus near the 6-o'clock meridian and into the lower fornix. Ophthaine was used as a local anesthetic. Tonography was performed 20 to 30 minutes after the injection, the injected eye being tested first. In the beginning many patients complained about immediate and late local irritation and pain. Later on we instilled suprarenin and cortisone after the test was over. No further patient complained about the late pain and only a few remarked upon a certain fullness of the eye. Pilocarpine was instilled whenever the intraocular pressure reached 35 mm. Hg or more after the test.

In the instances of combined Diamox-water-drinking test the tonometric readings were taken and the patient took 500 mg. of Diamox. At least one hour later the tension was again recorded and the water-drinking test was started. Thirty minutes later the final tonometric reading was taken and tonography was performed.

Results

The first charts illustrate the results of the water-drinking or the Priscoline test on normal eyes. As normal eyes are here grouped control eyes or eyes on which the subsequent clinical course did not substantiate the ori-

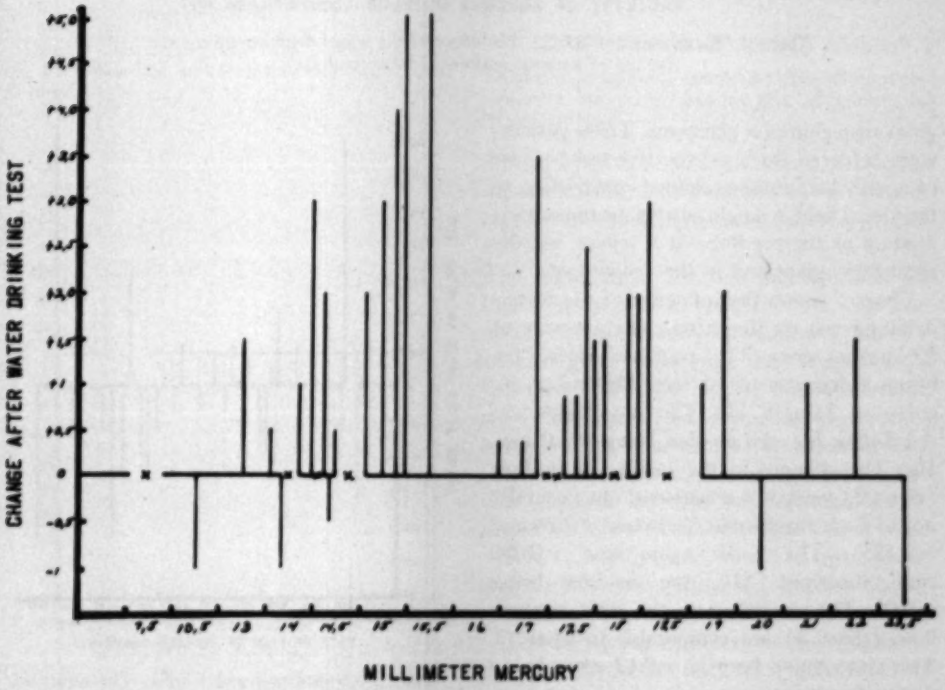


Chart 1 (Swanljung and Blodi). The effect of the water-drinking test on the intraocular pressure of 39 normal eyes.

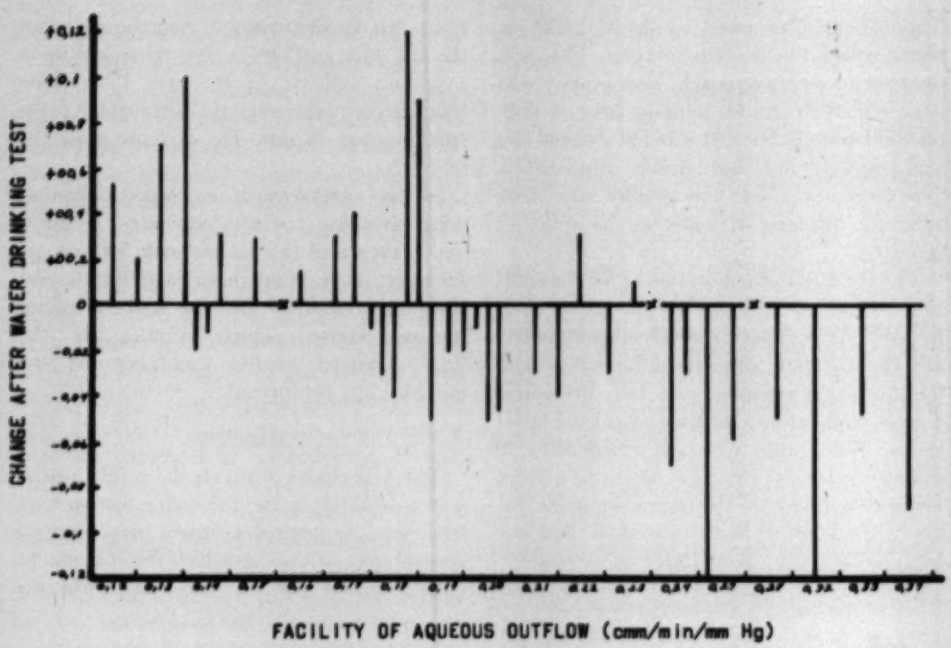


Chart 2 (Swanljung and Blodi). The effect of the water-drinking test on the facility of aqueous outflow of 39 normal eyes.

ginal suspicion of a glaucoma. These patients were referred for a provocative test because of a pale disc, an unexplained constriction of the visual field, a single suspicious tonometric reading or the presence of a severe, usually secondary, glaucoma in the fellow eye.

Chart 1 shows the influence of the water-drinking test on the intraocular pressure of 39 normal eyes. The response varied between a decrease of 1.5 mm. Hg and an increase of 5.0 mm. Hg. The mean value was +1.2 mm. Hg, the median being +1.0 mm. Hg. The changes in the facility of outflow (chart 2) were less consistent, but generally not of high magnitude (between +0.14 and −0.125). The mean value was −0.006 cmm./min./mm. Hg, the median being −0.01. The variations in the total aqueous flow (chart 3) are comparable to Chart 2. The mean value here is −0.12 cmm./min., the median being zero.

Charts 4 to 6 illustrate the reaction of 21

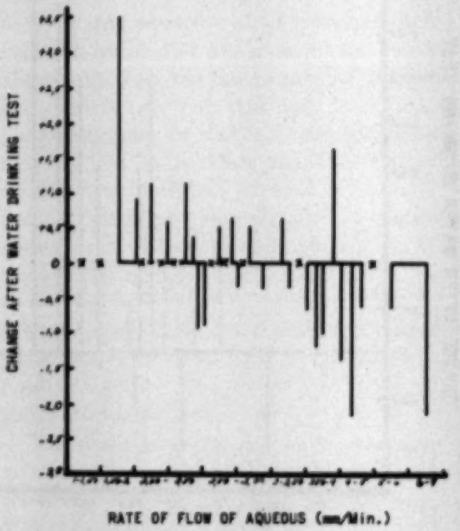


Chart 3 (Swanljung and Blodi). The effect of the water-drinking test on the rate of flow of aqueous of 39 normal eyes.

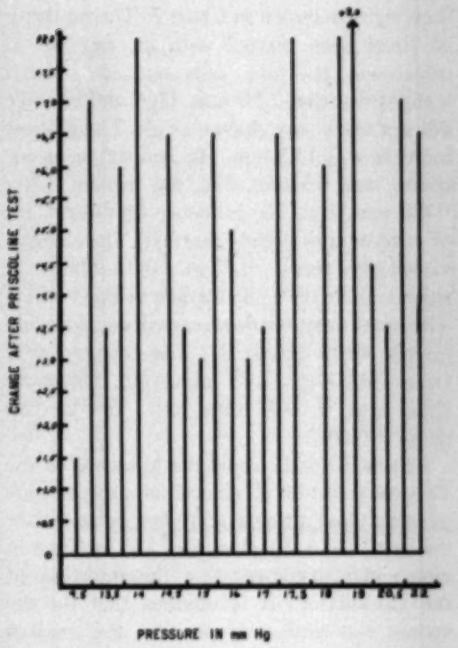


Chart 4 (Swanljung and Blodi). The effect of the Priscoline test on the intraocular pressure of 21 normal eyes.

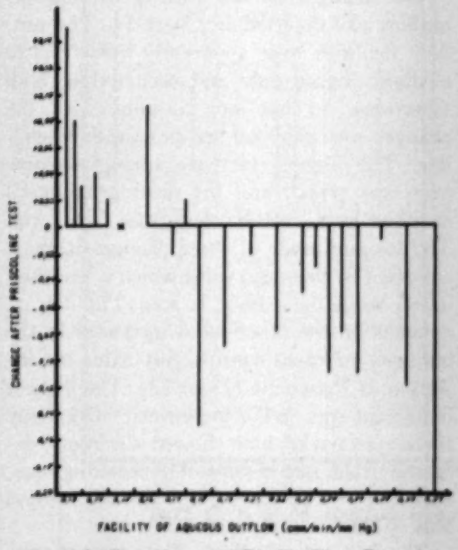


Chart 5 (Swanljung and Blodi). The effect of the Priscoline test on the facility of aqueous outflow of 21 normal eyes.

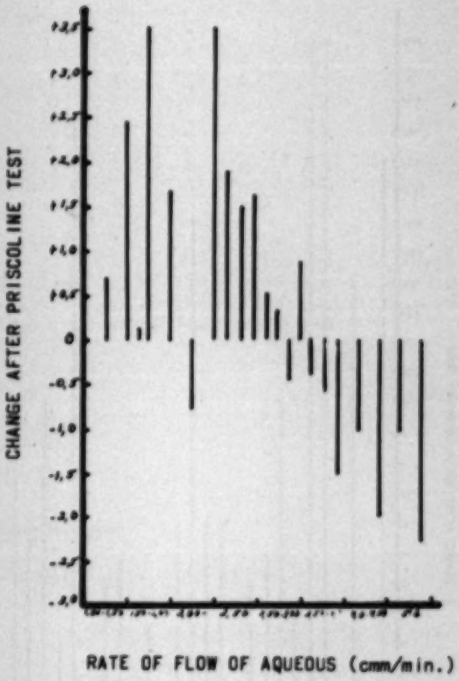


Chart 6 (Swanljung and Blodi). The effect of the Priscoline test on the rate of flow of aqueous of 21 normal eyes.

normal eyes to a subconjunctival Priscoline injection. This was followed by a constant rise in intraocular pressure (chart 4). This increase varied from 1.5 to 10.0 mm. Hg. The mean value for this increase was 5.8 mm. Hg, the median being 6.5 mm. Hg. The values for the facility of outflow (chart 5) showed great variations, the changes were between +0.15 and −0.21. The mean value was −0.03 cmm./min./mm. Hg, the median being −0.02. The values for the total rate of flow are similarly irregular (chart 6). The changes between the readings before and after injection of Priscoline varied from +5.30 to −2.23 cmm./min. The mean value was +1.0 cmm./min., the median being +1.25.

The following charts show the results of the two provocative tests on 21 glaucomatous eyes. Only such eyes were selected for this study which had no previous glaucoma sur-

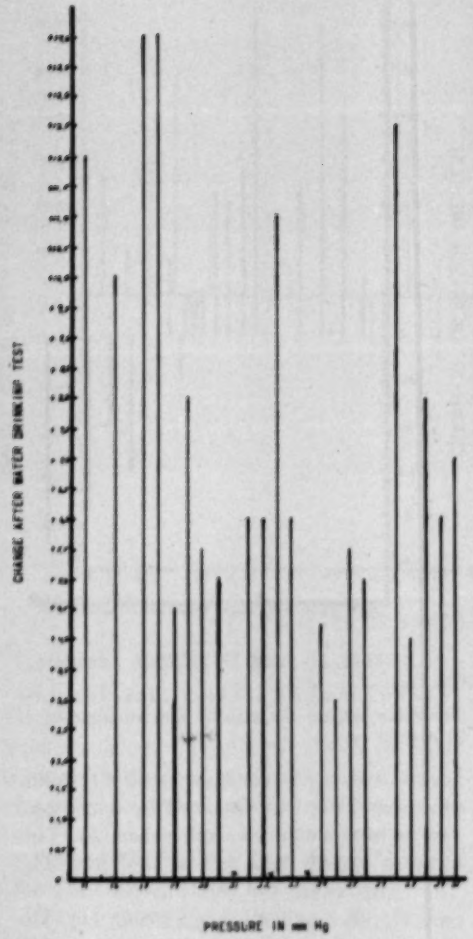


Chart 7 (Swanljung and Blodi). The effect of the water-drinking test on the intraocular pressure of 21 glaucomatous eyes.

gery and in which the intraocular pressure was not higher than 31 mm. Hg and only exceptionally higher than 28 mm. Hg. Some of the eyes were well controlled with a miotic. As all treatment was discontinued at least 24 hours before the provocative test, a few of these eyes had a pathologically increased pressure before the water-drinking test. No eye, however, was subjected to a Priscoline test with an initial pressure over 28 mm. Hg.

The effect of the water-drinking test on these eyes is shown in Chart 7. The majority of these eyes reacted with an increase in intraocular pressure; only one eye showed a slight decrease (1.0 mm. Hg) and one eye did not show any change at all. The highest increase was 13.5 mm. Hg and the mean increase was 6.5 mm. Hg, the median being +6.0 mm. Hg. The influence on the facility of outflow was slight (chart 8). The changes varied here from −0.05 to +0.07. The mean value was +0.009, the median being +0.015. The total rate of flow varied considerably in this series (chart 9). The changes were from +4.80 to −2.05 cmm./min. The mean value was +0.65 cmm./min., the median being +0.66.

Charts 10 to 12 show the influence of the Priscoline test on 10 glaucomatous eyes, nine of which had a tonography before and after the test. Chart 10 illustrates the increase in intraocular pressure after the injection of the vasodilator. It is obvious that the response was uniformly positive, the smallest increase being 6.5 mm. Hg and the highest 14 mm. Hg. The mean rise was 11.4 mm. Hg and the median was 11.50 mm. Hg.

The changes in the facility of aqueous outflow are depicted in Chart 11. The outflow facilities were apparently influenced to a slight degree only and occurred in both directions. In five out of nine eyes the changes were minimal and practically negligible. The highest increase among all nine eyes was +0.06 and the most pronounced decrease was −0.08 cmm./min./mm. Hg. The insignificance of these changes is characterized by the mean value which is less than 0.002, while the median is zero. The flow of aqueous on the other hand increased in the majority of cases considerably after the injection of Priscoline (chart 12). The highest increment was +4.0, the lowest −0.4. Only three eyes out of nine showed a minimal decrease in the rate of flow. The mean increase was here +1.02 cmm./min. and the median was +1.0.

The last set of charts illustrates the influence of Diamox on the water-drinking test in 12 glaucomatous eyes. Chart 13 gives the

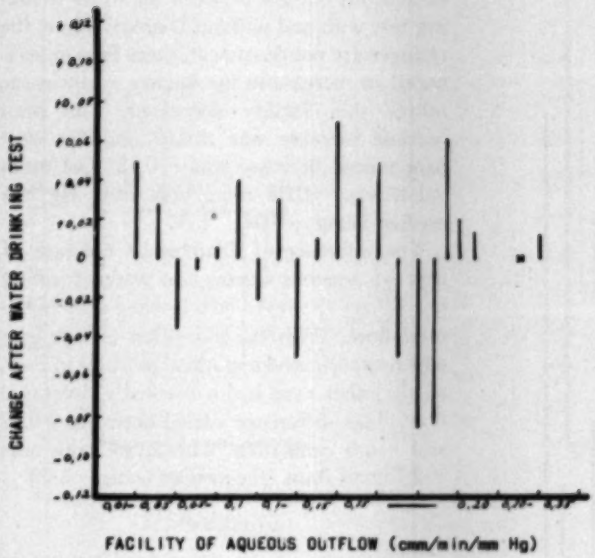


Chart 8 (Swanljung and Blodi). The effect of the water-drinking test on the facility of aqueous outflow of 21 glaucomatous eyes.

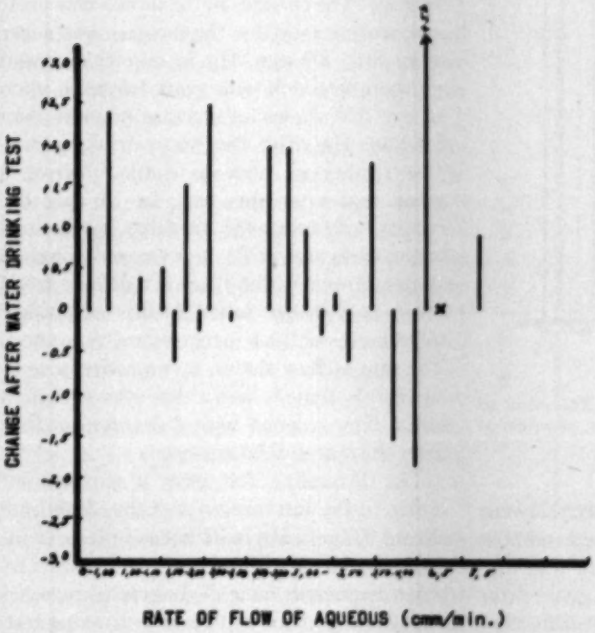


Chart 9 (Swanljung and Blodi). The effect of the water-drinking test on the rate of flow of aqueous of 21 glaucomatous eyes.

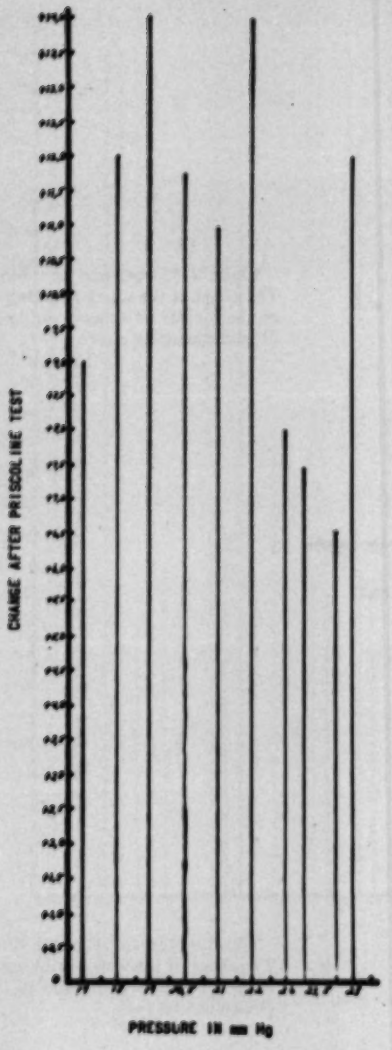


Chart 10 (Swanljung and Blodi). The effect of the Priscoline test on the intraocular pressure of 10 glaucomatous eyes.

difference in intraocular pressure between the water-drinking test with and without Diamox. The intraocular pressure was uniformly lower when Diamox was given prior to the water-drinking test. The difference varied from 17 to 1.5 mm. Hg. The mean drop was 9.5 mm. Hg and the median value was 8.75 mm. Hg.

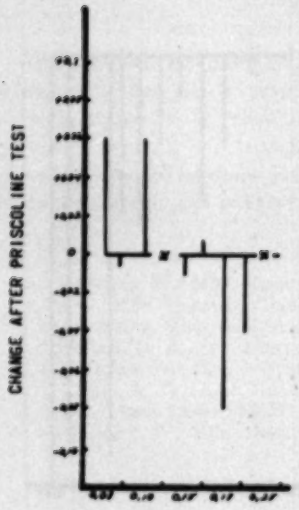
Chart 14 shows the difference in facility of aqueous outflow between the water-drinking test with and without Diamox. Here, the changes are not consistent, three eyes experienced an increase in the facility, while in the others this facility decreased. The most marked increase was +0.07 and the most pronounced decrease was −0.12. The mean value was −0.02 cmm./min./mm. Hg, the median being −0.04.

The influence of Diamox on the rate of flow of aqueous during the water-drinking test (chart 15) revealed a marked drop in the total flow. With the exception of one eye, which experienced a minimal increase in flow, all the other eyes had a markedly decreased flow. This difference varied between +0.02 and −6.0 cmm./min. The mean value was −2.7 cmm./min., the median being −1.09.

Comment

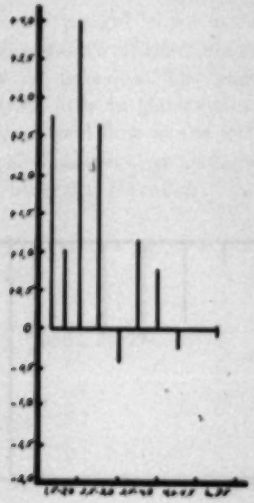
The results of the water-drinking test in normal eyes more or less confirmed previous findings. The changes in the intraocular pressure were erratic, but the increase was never more than 5.0 mm. Hg in our 39 eyes and we therefore look with great suspicion upon an eye that shows an increase of more than 6.0 mm. Hg after the water-drinking test. The facility of aqueous outflow varied a great deal after this test. In 13 eyes the facility was increased, in three it remained unchanged, and in 23 it decreased. Chart 2 shows, however, that there is a definite trend for eyes with low initial facility of outflow to respond with an increase and vice versa. The rate of flow shows, by necessity, a similar distribution. It seems that eyes with high initial flow respond with a decrease in flow after the water-drinking test.

The Priscoline test gave a uniform increase in the intraocular pressure. It did not exceed 9.0 mm. Hg in 21 normal eyes. An increase of 10 mm. Hg or more is therefore of some diagnostic value. Tonography revealed a response similar to the water-drinking test. There was a great variation in the facility of outflow with a definite tendency for the eyes with high initial facility to respond with



FACILITY OF AQUEOUS OUTFLOW (cmm/min/mm Hg)

Chart 11



RATE OF FLOW OF AQUEOUS (cmm/min.)

Chart 12

Charts 11 and 12 (Swanljung and Blodi). The effect of the Priscoline test on the facility of aqueous outflow and the rate of flow of aqueous of nine glaucomatous eyes.

a marked decrease. It seems that the pronounced hyperemia produced with Priscoline does not increase the total rate of flow when the initial flow is high.

In the 24 glaucomatous eyes the water-drinking test produced a uniform increase in the intraocular pressure, which was 6.0 mm. Hg or more in 18 eyes. The facility of outflow varied comparatively little and the changes that occurred did not follow any particular pattern. This would indicate that the usually decreased facility of outflow in glaucomatous eyes is little influenced by this provocative test. The total rate of flow showed an increase in the majority of cases. The six exceptions to this increase occurred in those eyes which had a minimal increase in intraocular pressure.

The Priscoline test produced a uniform increase in intraocular pressure in 10 glaucomatous eyes. In six of these eyes the increase was more than 10 mm. Hg. Four of these six eyes had previously given a negative result to a water-drinking test. The changes in the facility of outflow were small and inconsistent. The total rate of flow increased in the majority of cases.

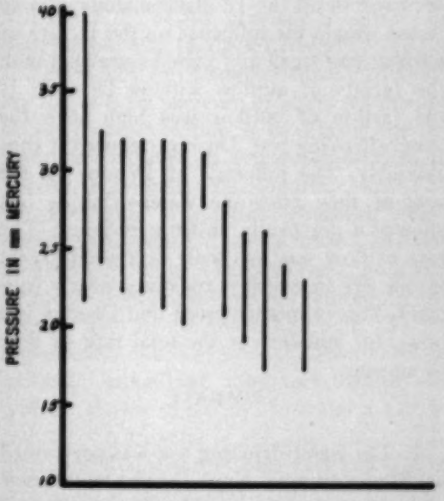
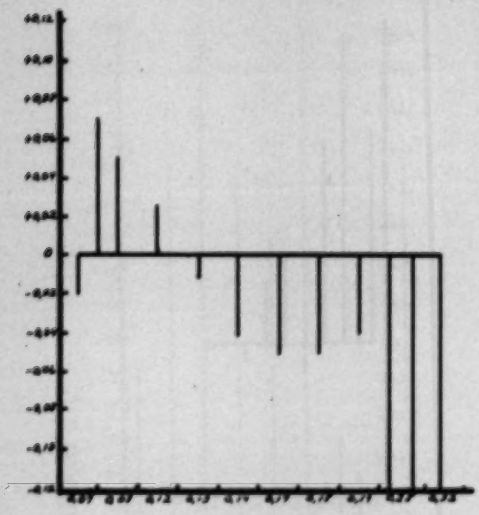


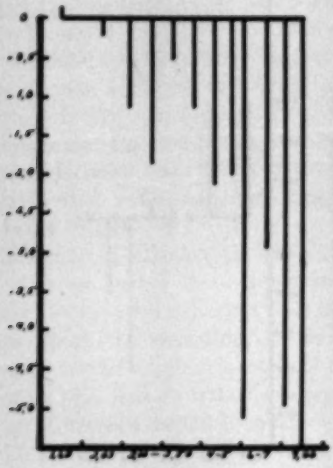
Chart 13 (Swanljung and Blodi). The influence of Diamox on the intraocular pressure after a water-drinking test in 12 glaucomatous eyes.

The influence of Diamox on the water-drinking test was first of all a decrease in the



FACILITY OF AQUEOUS OUTFLOW (cmm/min/mm Hg)

Chart 14



RATE OF FLOW OF AQUEOUS (cmm/min.)

Chart 15

Charts 14 and 15 (Swanljung and Blodi). The influence of Diamox on the facility of aqueous outflow and on the rate of flow of aqueous after a water-drinking test in 12 glaucomatous eyes.

response of the intraocular pressure. This occurred in all the 12 glaucomatous eyes so tested. Again the influence on the facility of outflow was small and varied somewhat with the facility of outflow without Diamox. If the facility of outflow was high after the water-drinking test, Diamox reduced it considerably. The influence of Diamox on the rate of flow after the water-drinking test showed a practically uniform response. The rate of flow was markedly decreased except in one eye in which it remained nearly constant. This is another proof that Diamox is a powerful inhibitor of the total rate of flow of aqueous.

Summary

1. The water-drinking test was performed on 39 normal eyes. None showed an increase of more than 5.0 mm. Hg. The facility of outflow varied considerably but seemed to increase when the initial facility of outflow was low and vice versa. In the same way eyes with an initial high rate of flow responded with a decrease in flow and vice versa.

2. The Priscoline test was done on 21 normal eyes. All experienced an increase in intraocular pressure up to 9.0 mm. Hg. Facility of outflow and toal rate of flow of aqueous behaved practically identical as in the water-drinking test.

3. The water-drinking test was done on 24 glaucomatous eyes. Six eyes did not respond with an increase in the intraocular pressure of 6.0 mm. Hg or more. The facility of outflow changed very little. The rate of flow increased in all cases with a pathologic response of the intraocular pressure.

4. The Priscoline test was performed on 10 glaucomatous eyes, six of which experienced a rise in intraocular pressure of more than 10 mm. Hg. The facility of outflow did not change considerably. The rate of flow was markedly increased.

5. A dose of 500 mg. Diamox was given one hour before the water-drinking test in 12 patients with chronic wide-angle glaucoma. The rise in intraocular pressure was uniformly reduced. The facility of outflow showed inconsistent changes. The total rate of flow was decreased.

Conclusions

The water-drinking test should be the first provocative test in a suspicious case of chronic glaucoma. It reliability was, in our series, 70 percent. If it is negative, the Priscoline test should be done. Its reliability was 60 percent but it was positive in cases which did not respond to the water-drinking test. Both tests are effective via an increased total flow of aqueous. The facility of outflow changes little in glaucomatous eyes. The increased total flow in the water-drinking test can be diminished by Diamox.

University Hospitals.

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IONOMETRY OF THE AQUEOUS HUMOR*

Report on the mode of action of acetazoleamide

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According to Friedenwald and Kinsey's[19] well-known formula

$$[OP = \frac{k_{in}}{k_{in} + k_{out}}[OP_{Aq} - OP_{Pl} + Cap\ P],$$

the smaller the facility of outflow of the aqueous humor (k_{out}) and the larger the value of the fraction $\frac{k_{in}}{K_{in}+K_{out}}$, the greater will be the influence which eventual variations in the mean uveal capillary pressure (Cap P) and in the osmotic pressure of blood plasma (OP_{Pl}) and aqueous (OP_{Aq}) will have on the intraocular pressure (IOP). When as in the normal human eye, that fraction is equal to 0.1, relatively large variations of Cap P, OP_{Pl} and OP_{Aq} will determine but slight changes in intraocular pressure; but when, as in glaucoma, the outflow is impaired and the value of $\frac{k_{in}}{k_{in} + k_{out}}$ approaches 1, any variation in the magnitude of those factors will affect the ocular pressure in a tremendous proportion (Urrets-Zavalia[26]).

This is why (1) all agents capable of altering the osmotic pressure of blood plasma are by far more operative in modifying the intraocular pressure in glaucomatous than in normal individuals (de Roetth[22]), why (2) the variations in capillary pressure which appear in response to phasic diurnal changes in arteriolar tonus (Duke-Elder[7]) give rise to much larger tensional oscillations in the former than in the latter, and why (3) the hypotensive effect of cyclodiathermy and of carbonic anhydrase inhibitors—which, as will be shown presently, provoke a fall in the osmotic pressure of the aqueous—is very pronounced in glaucomatous eyes and almost negligible in normal eyes.

Of the various factors upon which the intraocular pressure depends, the facility of outflow may be easily determined in almost all cases by means of tonographic records and expressed in terms of a coefficient, C, representing the rate at which fluid can be squeezed out of the eye in cubic millimeters, per

* From the Instituto de Investigaciones Médicas "Mercedes y Martín Ferreyra" and the Clínica del Glaucoma y Cirugía de Córnea (Hospital Italiano).

minute and per millimeter (mercury) pressure (Grant [14-16]). The resistance to outflow may also be measured by means of a special technique developed by Goldmann[11] on different grounds and expressed in terms of what has been called by him *scheinbarer Abflussdruck*. On the other hand, the uveal capillary pressure cannot be measured clinically except in the rare cases where some capillaries are visible biomicroscopically on the anterior surface of the iris (Fritz[10]). The normal osmotic pressure of the aqueous humor and the changes which it may experience under different circumstances have been studied only exceptionally, in experiments designed to corroborate the fact that the aqueous humor is hypertonic with respect to the plasma and that this hypertonicity results from the activity of the cillary body.

Up to the time of writing and to the best of our knowledge, however, no formal attempt has been made to appreciate the modifications which this pressure could eventually undergo through the influence of certain ocular operations or of the administration of drugs.* The osmotic pressure of the aqueous, which normally exceeds that of the plasma ($OP_{Aq} > OP_{Pl}$), does not seem to be substantially modified in chronic glaucoma; yet the hypothesis that some therapeutic agents much used in glaucoma—such as the cyclodiathermy operation or the administration of acetazoleamide—owe their hypotensive effect to an inhibition of the secretory function of the ciliary epithelium and to the subsequent impoverishment of the aqueous in osmotically active salts (Urrets-Zavalia[25]), suggests that the measurement of the osmotic pressure of that fluid might possess an immediate clinical value, in so far as it would represent a practical means of assessing the effectiveness of treatments which do not bring about changes in the facility of outflow, just as tonography does with respect to those which produce such changes.

As direct estimates of the osmotic pressure or of the freezing point of such small samples of aqueous humor as can be obtained in clinical and even in experimental work are difficult and require expensive equipment, it has been considered preferable to study the conductivity of this humor by appraising the resistance which a given column of the same opposes to the passage of an alternating current, and to calculate, from the variations registered, the changes in the total concentration of dissociated salts which must have taken place. It will be remembered that the power of conducting a current depends for a fluid on the actual number of ions engaged in the carriage of the charges and also on the rate at which they move (Duke-Elder[6]). Hence the name of ionometry which, short of a more precise designation, was given to the procedure involved.

Recently, Duke-Elder, Davson, and Maurice[8] made use of an apparatus, similar in many respects to the one to be described, in order to detect the changes in conductivity experienced by the aqueous humor and plasma when one fluid was dialyzed against the other across a separating collodion membrane.

Instrument

The conductivity cell is in the form of a pipette of 0.05-cc. capacity which bears in each extremity a platinum wire electrode secured into place by an airtight plexiglas cap. The pipette, which is filled by suction without the aqueous being exposed to the escape of carbon dioxide, is immersed in water contained in a thermally insulated box, and kept at a temperature of 37 ± 0.2°C. by means of a thermostat. The electrodes are connected to a modified Wheatstone bridge, equipped with high-precision resistances and maintained at constant room temperature. Readings in ohms are made of the resistance represented by the fluid interposed between the electrodes.

* A succint preliminary report on the subject, presented to the Sociedad de Oftalmología de Córdoba on December 10, 1954, has already been published (Arch. oftal. Buenos Aires, 30:92-93 [Feb.-Mar.] 1955).

In order to express directly the results in terms of variations in molar concentration, the instrument was calibrated through a series of readings made of a number of sodium chloride solutions of different titers; the results were plotted on a chart and a curve was drawn, which permitted ascertaining at a glance the significance of any change in resistance and to formulate it in mM/L. equivalent of sodium chloride.

As duplicate readings on any solution of a molarity ranging from 0.1,300 to 0.1,500 were consistently found to agree to one part in 200, it was assumed that an actual difference in ionic concentration existed whenever a larger discrepancy was encountered. It is to be noted that Duke-Elder, Davson, and Maurice[8] were able to make measurements of a precision of no less than one part in a thousand, a performance which we have been unable to match, for in spite of all efforts repeatability could not be carried beyond the half-percent limit.

Method

The experiments designed to demonstrate the mode of action of acetazoleamide (Diamox*) were performed on dogs, anesthetized with diallylbabituric acid (Dial®) given intraperitoneally at a dose of 0.06 gr./kg. body weight. Thirty minutes later, a drop of one-percent tetracaine solution was instilled onto the cornea of one eye and 0.05 to 0.15 cc. of aqueous humor was removed by means of an Amsler cannula. The conductivity of the sample was measured and a solution of the sodium salt of acetazoleamide in bidistilled water injected at a dose of 50 mg./kg. body weight. After a delay of one to two hours, which allowed the aqueous humor to be totally renewed, an equal amount of fluid was withdrawn from the fellow eye and the conductivity measurement was repeated.

Results

Seven dogs were submitted to the procedure just outlined. In five others, intended as controls, the experiment was identically performed but bidistilled water was substituted for the sodium-acetazoleamide solution.

In none of the control animals did the conductivity show any change which could not be considered negligible (table 1). This was also the case in one of the treated animals, in which acetazoleamide was given intraperitoneally and the aqueous sample obtained after a delay of only an hour. On the other hand, in the remaining six animals of this group, in which the drug was administered intravenously and a delay of two hours allowed before the second eye was tapped, a net fall in conductivity was recorded (table 2), which corresponded to an average decrease in osmotic pressure of no less than 5 mM/L equivalent of sodium chloride.

When the mean variation in resistance of the one series, as stated in percentages, 3.16 $\pm$ 2.11, is adequately compared to that of

* Diamox (brand of 2-acetylamino-1,3,4-thiadiazole-5-sulfonamide) was supplied through the courtesy of Dr. F. C. Ottati, Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York.

TABLE 1

CHANGES IN ELECTRICAL RESISTANCE OF AQUEOUS HUMOR (ΔRAq) FOUND WHEN READINGS WERE TAKEN FROM CONTROL DOGS INJECTED WITH BIDISTILLED WATER

Experiment No.	Route of Injection	Interval between Injection and Measurement	ΔRAq %
1	Intravenous	2 hr.	0.00
2	Intravenous	2 hr.	+1.10
3	Intravenous	1 hr. 45 min.	+0.70
4	Intravenous	2 hr.	−0.20
5	Intravenous	2 hr.	−0.60

Average ΔRAq: +0.20 $\pm$ 0.69%.

TABLE 2

CHANGES IN ELECTRICAL RESISTANCE OF AQUEOUS HUMOR OF DOGS (ΔRAq) AFTER ADMINISTRATION OF SODIUM ACETAZOLEAMIDE

Experiment No.	Route of Injection	Interval between Injection and Measurement	ΔRAq %
1	Intraperitoneal	1 hr.	0.00
2	Intravenous	2 hr.	+6.20
3	Intravenous	2 hr.	+4.20
4	Intravenous	2 hr. 10 min.	+3.10
5	Intravenous	2 hr.	+4.60
6	Intravenous	1 hr. 55 min.	+2.90
7	Intravenous	2 hr.	+1.10

Average ΔRAq: $+3.16 \pm 2.11\%$.

the other, 0.20 ± 0.69, in order to ascertain the consequence of the changes registered, it is seen that the difference, 2.96 ± 0.63, being equal to 4.69 times its own probable error,* may be contemplated as statistically significant. Accordingly, the fall in conductivity presented by the aqueous humor of those animals which received an injection of acetazoleamide is attributed to a reduction in its electrolyte content. This, in turn, seems dependent upon the effect of the drug on the secretory function of the ciliary epithelium and upon the establishment of as nearly a perfect thermodynamic equilibrium between this fluid and the blood plasma as would be permitted by the flow still occurring through the anterior drainage channels.

COMMENT

A high concentration of bicarbonate and of sodium in the aqueous humor has been reported recently by Kinsey,[20] whose findings substantiate Friedenwald's[9] theory of an active transfer of electrolytes into the eye by the ciliary epithelium and corroborates Duke-Elder, Davson, and Maurice's[8] contention that the excess of osmotic pressure in the aqueous humor as compared with the plasma—a difference which occurs in the opposite sense from that required by Donnan equilibrium—is due to an excess of electrolyte in the former.

Although, as pointed out by Becker,[2] carbonic anhydrase has not yet been directly implicated in this transfer, the existence of an extremely potent carbonic anhydrase inhibitor, acetazoleamide, seemed to warrant an exploration of its effect on the intraocular pressure. The opinions on the effect of this drug on normal eyes are by no means unanimous; on the whole, however, it may be safely assumed that it is unable to influence significantly the intraocular pressure, while it has been shown (Becker[4]) to cause a 55 to 65 percent suppression of the aqueous outflow. This must be attributed to the previously noted fact that, the fraction $\frac{k_{in}}{k_{in} + k_{out}}$ being very small in the normal, such changes as could affect the osmotic forces of the aqueous and plasma can have but a small influence on the ocular pressure. Yet, it would be ill advised to infer from this that the latter is normally not dependent upon the carbonic anhydrase activity of the anterior uvea and upon the electrolytic content of the aqueous; the truth is that the intraocular pressure is actually dependent upon the activity of this ferment, though not on this activity alone, but on other factors as well, among which

* The probable error $\varepsilon\Delta$ of the difference $(m_1 - m_2)$ encountered between both averages was estimated by means of the formula $\varepsilon\Delta = \pm\sqrt{\varepsilon_1^2 + \varepsilon_2^2}$, where ε_1 and ε_2 are the probable errors of the two mean results.

To determine the probable error of a given mean, the equation $\varepsilon = \pm 0.6745 \frac{\sigma}{\sqrt{n-1}}$ was used, where σ stands for the standard deviation of a single observation and n for the total number of observations performed.

that represented by the bulk flow of the aqueous is highly significant.

On the other hand, the collected evidence shows that the drug has a definite hypotensive action in most cases of primary or secondary glaucoma, that this action is all the more marked the smaller the facility of outflow becomes and the larger the moment which the factors OP_{Aq} and OP_{Pl} proportionally acquire in the maintenance of intraocular pressure, and that this action is independent of its renal effect and of the associated alterations in circulating electrolytes (Becker[3]).

The presence of carbonic anhydrase in the ciliary body and the iris has been demonstrated by Westrand,[28] and the inhibitory effect of acetazoleamide on the activity of this ferment as present in the uvea has been determined on tissue extracts by Green, Capper, Bocher, and Leopold,[18] who presumed that carbonic anhydrase might be directly involved in the formation of the bicarbonate ions found in the intraocular fluid. Were this enzyme inhibited by acetazoleamide, sodium and bicarbonate would enter the aqueous in a lesser amount and osmotic pressure drop, the outcome being a lesser water inflow and a fall in intraocular pressure. Notwithstanding, as pointed out by Breinin and Görtz,[5] in the absence of electrolyte determinations this scheme had to remain conjectural, however reasonable and in accord with what is known of acetazoleamide action it appears.

As the increment in aqueous resistance which was found by us after acetazoleamide administration must necessarily correspond to a markedly decreased electrolyte content, and as it is hard to accept that ions other than bicarbonate and sodium might be implicated in this fall, one is almost forced to admit that the reaction is dependent on an actual inhibition of carbonic anhydrase, for it seems improbable that no connecting link should exist between the facts that (1) acetazoleamide inhibits the carbonic anhydrase present in the anterior uvea and (2) that acetazoleamide leads to a fall in the electrolyte content of the aqueous humor. Nevertheless, Green, Bocher, and Leopold's[17] direct determinations of bicarbonate ion concentration apparently disprove the assumption that intravenous injection of acetazoleamide lowers appreciably the bicarbonate ion content of the aqueous. Their results are, however, not absolutely conclusive, in that there appears to be an imperfect agreement between some of them, and they require confirmation.

Besides, it must be remembered that the secretory function of the anterior uvea is only partly necessary for the maintenance of the bicarbonate concentration of the aqueous, a process of simple diffusion being responsible for the major part of this ion's content, and that, accordingly, only minor changes in the latter might be expected to occur even after a complete abrogation of that active transfer.

Although the composition of the aqueous humor varies in the different species in so far as the relative distribution of sodium, bicarbonate, and chloride ions is concerned, it may be presumed that the explanation which the herein reported experiments suggest for the effect of acetazoleamide in dogs is also valid for the reaction as observed in man.

The supposition advanced by Green, Bocher, and Leopold[17] that the reduction in the bicarbonate content of the anterior aqueous humor of the rabbit, which was found by them after a subconjunctival injection of acetazoleamide, is due to an augmented permeability of the blood-aqueous barrier and to a dilution of the aqueous by diffusion, require special consideration. In fact, a moderate increment in the permeability of the barrier and in the protein content of the aqueous humor does occur in that case, as shown by these authors, as it does following oral (Appelmans and Michiels[1]) or intravenous (Urrets-Zavalía and Remonda[27]) administration of the drug. However, it is doubtful whether capillary dilation and increased permeability actually result in a drop in the electrolytic content and in the osmotic pressure of the aqueous, either in

this case or in others where a veritable collapse of the barrier is known to occur in response to noxious or pharmacologic influences. Indeed, Linnér and Friedenwald's[21] studies on the appearance time of fluorescein through the pupil into the anterior chamber do not seem to support the idea that the increase in the permeability of the blood-aqueous barrier plays a role in the mechanism whereby acetazoleamide reduces the intraocular pressure.

It is well known that the short-term use of acetazoleamide produces no tonographically measurable changes in the facility of outflow (Becker,[2,4] Breinin and Görtz[5]). Therefore, the decrease observed in the rate of aqueous outflow (*minute volume, débit*) must be a consequence of a reduced water inflow.

The mechanism through which acetazoleamide lowers the intraocular pressure is in all probability similar or identical to that involved in the case of cyclodiathermy (Urrets-Zavalía[25]), of retrobulbar procaine injection (de Roetth and Carroll[23]) and of that *Sekretionsstop* observed by Goldmann[12,13] for many months after the performance of a cyclodialysis or an antiglaucomatous iridectomy. As pointed out previously (Urrets-Zavalía[26]), this stop of secretion must actually be looked on as an arrest in the conveyance of osmotically active salts by the ciliary epithelium, and not as a total detention of the water inflow.

In passing, it is convenient to note that one cannot subscribe to the simplified point of view that the rate of outflow of the aqueous humor is necessarily equal to its rate rate of formation, since it is known that the former may eventually become insignificant, or even null, without the inflow of water being more than moderately delayed. Such is the case when the true outflow pressure—defined by Goldmann[11] (wirklicher Abflussdruck) as the difference between the intraocular pressure and the pressure in the episcleral veins—becomes smaller than the resistance to outflow as a result either of trauma or disease, or surgical interference, or of some therapeutic agent. In these circumstances, the aqueous veins happen to be filled with blood and are no longer distinguishable; the eye becomes a watertight system in so far as the drainage apparatus is concerned and the water and solute exchanges proceed, though under modified gradients and in a different magnitude, through the blood-aqueous barrier (Urrets-Zavalía[24]).

Summary and Conclusions

A fall in the electric conductance of the anterior aqueous humor of the dog was found to occur after intravenous injection of acetazoleamide.

This fall, which corresponds to a drop in the electrolyte content and the osmotic pressure of the aqueous of an order of 5 mM/L equivalent of sodium chloride, seems to account for the hypotensive effect of the drug on glaucomatous eyes.

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USE OF ACETAZOLEAMIDE (DIAMOX)*

FOR ENDOTHELIAL CORNEAL DYSTROPHY AND DISEASED CORNEAL GRAFTS

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In 1952, a successful corneal transplant was done on a 40-year-old woman with keratoconus. The graft remained perfectly clear for 18 months. Vision in the operated eye stabilized at 20/30. At that time corneal edema appeared. With the biomicroscope one could then observe degeneration of the corneal endothelium. For some time hypertonic solutions and cortisone improved the function but the edema gradually became more pronounced and finally would no longer respond to any treatment. It was then decided to try Diamox. The patient was given 500 mg. Diamox and was re-examined after two hours. The corneal edema had all but disappeared. Vision had improved from 20/400 to 20/40. Encouraged by this dramatic result, Diamox was used in other cases of endothelial dystrophy. Some of these were primary Fuchs' type dystrophies and others had developed secondary to corneal grafting. Table 1 shows the response to the Diamox treatment in each case.

It should be emphasized that glaucoma

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TABLE 1

EFFECT OF ORALLY ADMINISTERED DIAMOX ON CORNEAL EDEMA AND VISUAL ACUITY IN PRIMARY AND SECONDARY ENDOTHELIAL DYSTROPHY (22 Cases)

Two Hours After Oral Administration of 500 mg. Diamox				
	Corneal Edema		Visual Acuity	
	Improved	Not Improved	Improved	Not Improved
Number of Cases	18	4	14	8

was present in none of these cases. Admittedly, in only a comparatively few instances was the intraocular pressure measured with the tonometer. In accordance with Doggart[1] I feel that, in Fuchs' dystrophy, considerable harm may be done by frequent instrumental tonometry.[7] Since corneal edema rarely ever develops until the intraocular pressure has reached a level easily recognizable by an experienced clinician through palpation, tonometry was considered inadvisable.

Table 1 demonstrates that in 18 of 22 cases the corneal edema had subsided and in 14 of 22 cases the visual acuity had improved materially. Most of the cases which showed clearing of the cornea without improvement of vision were complicated by other disturbances—in particular, beginning cataracts.

The degree of visual improvement in the individual cases is seen from Table 2. While not all patients responded to treatment with Diamox as dramatically as did the first-mentioned case, the improvement was quite remarkable in over half of them. The question arises: for how long a period of time can the effect of Diamox be maintained? In the first-mentioned case, the treatment is still effective after 13 months. In another case, Diamox has been effective for 11 months. As a matter of fact, all cases in which corneal clearing could be demonstrated within two hours after the initial test dose continued to respond to the drug during the entire period of observation.

TABLE 2

VISION BEFORE AND TWO HOURS AFTER ORAL ADMINISTRATION OF 500 MG. DIAMOX IN THOSE PATIENTS WHO DID RESPOND

Patient No.	Eye	Vsual Acuity Before Diamox	Visual Acuity After Diamox
3	R	20/60	20/40+
3	L	20/70	20/40+
4	R	20/200	20/50+
5	L	20/400	20/200+
6	R	20/50	20/40
6	L	20/200	20/100
8	R	20/200	20/100
8	L	20/400	20/80
9	R	20/200	20/70+
12	R	20/70	20/40
12	L	20/70	20/40
13	R	20/40	20/30
13	L	20/400	20/70
16	L	20/400	20/40

The effective dose of Diamox varies from case to case. In general it may be said that relatively small and infrequent doses are required. In no case was a maintenance dose exceeding 250 mg. Diamox per day necessary. Most cases do well with 250 mg. every other day. Patients who are good observers are able to determine without supervision how often the drug needs to be taken. They are allowed to work out the minimum requirement for satisfactory visual results.

Table 3 explains how one patient was able to space the intake of Diamox according to her need over a period of seven months. At one time the cornea remained clear for a month without Diamox.

In some cases it was observed that, when Diamox was discontinued, the edema returned and the vision failed again only to improve promptly when the treatment was resumed.

TABLE 3

INTERVALS AT WHICH ONE PATIENT HAD TO TAKE 250 MG. DIAMOX TO KEEP HER CORNEA CLEAR OVER A PERIOD OF SEVEN MONTHS

Month	Days of the Month on Which Diamox was Taken
August	1, 4, 5, 8, 10, 11, 13, 14, 18, 21, 25
September	1, 4, 7, 11, 15, 18, 19
October	None
November	22, 25
December	10, 15, 17, 28
January	2, 5, 6, 9

Among the circumstances that seem to influence the need for Diamox, atmospheric humidity was definitely mentioned by one patient. He asserted that vision diminished on humid sultry days. This observation may be explained by the difficulty with which the fluid evaporates from the cornea in times of high humidity.

Since the treatment of endothelial dystrophy with Diamox may be extended over an indefinite period of time, the question arises as to whether this can be done without risking damage to the general health of the patient.

Contrary to the requirements in glaucoma, where around-the-clock administration is necessary, the dosage for endothelial dystrophy approximates that used in cardiac edema —250 mg. daily or every other day. Since this type of dosage allows for a diurnal acid-base recovery period during which the blood bicarbonate may be repleted, harmful effects seem to be unlikely. Yet, because Diamox is a sulfonamide, particular attention should be paid to the possible development of agranulocytosis. I was able to find only one case of agranulocytosis following daily administration of 250 mg. Diamox reported in the literature (Pearson, Binder, and Neber[2]).

Blood studies in my cases in which the drug was used for prolonged periods have always revealed normal pictures. Nevertheless, such examinations should be carried out from time to time. Such side effects as parasthesias, anorexia, drowsiness, and so forth have been reported by Becker,[3] Grant and Trotter,[4] and others as occurring rather regularly during the treatment of glaucoma with Diamox. In my series, evidently due to the small and infrequent doses, these side effects have been rare and so negligible that it was never necessary to discontinue the drug.

As to an explanation of the clearing effect of Diamox in endothelial dystrophy, the following possibilities may be considered:

1. The corneal edema in endothelial dystrophy is due to the fact that the endothelial layer has become permeable to the aqueous humor. Leber[5] has demonstrated that the rate of penetration of fluid through Descemet's membrane when denuded of endothelium is dependent to some extent on the hydrostatic pressure to which it is exposed.

Vogt[6] suggested reducing the intraocular pressure to subnormal levels by means of filtering operations and reported favorable results with this method. Unfortunately, I have not been able to confirm Vogt's observations nor have I found any other favorable reports in the literature.

It is unlikely that the intraocular pressure was markedly reduced after the administration of Diamox in the nonglaucomatous eyes of the group under present consideration. As already mentioned, not many instrumental measurements of intraocular pressure were performed. Tonometric examination in two cases showed the pressure to be 16 mm. Hg before and 16 mm. Hg after Diamox in one instance and 14 mm. Hg before and 13 mm. Hg after Diamox in another instance. This observation is in keeping with the results reported by Becker[3] and Grant and Trotter[4] who also found very little change in the intraocular pressure of normal eyes after Diamox. The reduction in intraocular presure cannot, therefore, be the cause of the clearing effect.

2. A second possibility is that the clearing of the cornea may be directly connected with the diuresis caused by Diamox. The diuretic effect of Diamox is connected with increased excretion of sodium. Since the blood-plasma levels of sodium are found to be about normal in spite of the increased output, sodium ions must pass into the blood plasma from the tissues. This means a relative increase in the osmotic pressure of the blood as compared with the tissues. Consequently, water will pass from the tissues into the blood stream. This dehydrating effect may clear the cornea.

3. Since, according to Friedenwald and others, the effect of Diamox in glaucoma is to be found in a direct action on the mechanism of secretion of the aqueous humor, a

third possibility should be considered in a direct effect of the carbonic anhydrase inhibitor on the metabolism of the cornea. One way of answering this question was by exclusion:

If another diuretic, acting on another principle, would produce the same result as Diamox there was no need for the supposition of a specific action on the cornea. The mercurial diuretic, Mercuhydrin, was chosen for comparison. Mercurial diuretics also produce an increased excretion of sodium but through a different mechanism than the carbonic anhydrase inhibitor, presumably impairing the reabsorption of sodium through a toxic effect on the tubular epithelia of the kidney. Six patients were given one cc. of Mercuhydrin intramuscularly and two and one-half hours later the effect on the cornea was studied.

In one case Mercuhydrin produced about the same clearing effect as Diamox. In two cases Mercuhydrin had some effect but not as much as Diamox. Two cases which previously had responded to Diamox did not respond at all to Mercuhydrin. One case which had not been improved by Diamox did not benefit from Mercuhydrin. These results being somewhat inconclusive, further studies are necessary to ascertain the exact mechanism which is involved.

The treatment of endothelial dystrophy is often disappointing. It is felt, therefore, that any addition to the therapeutic armamentarium of this disease which promises some improvement in vision, even if only for a certain period of time, is worth trying. With this in mind, this clinical study is reported.

Summary

The clearing effect of Diamox on the corneal edema in cases of primary and secondary endothelial dystrophy was reported.

Visual acuity could be substantially improved in most of the cases.

Relatively small and infrequent doses were required for the maintenance of visual improvement for indefinite periods.

Side effects were rare and negligible and never necessitated the discontinuation of the drug.

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PRIMARY GLAUCOMA: ETIOLOGY AND GENERAL CONSIDERATIONS*

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Plus ça change, plus c'est la même chose.
—French Proverb

INTRODUCTION

During the symposium held on glaucoma at the XIII International Congress of Ophthalmology, 1929, Sir Stewart Duke-Elder closed his remarks on the etiology and nonoperative treatment of glaucoma with the following words:

"On the whole the final word which must be said both regarding the etiology and the treatment of glaucoma is that we do not know. The crux of the matter is physico-chemical, and some day we can but hope that it may be elucidated by physico-chemical means. The whole of the science of medicine and the art of surgery are slowly becoming advanced exercises in applied physical science and when we as physicians fail to cure and resort as surgeons to palliative procedures, we are merely admitting the problems which confront us are too complicated for us to understand, our material too complex for us to manipulate, and our knowledge as yet too fragmentary and inadequate for us to apply it systematically."

Twenty-five years later we must confess that our knowledge regarding this widespread, blinding disease is still very scanty. It is, however, fair to say that these years have witnessed a painfully slow accretion of accurate information, particularly along the physico-chemical lines prophesied by Duke-Elder. I believe we have reason to be optimistic about the more rapid solution from now on of many of the problems that are part of the whole complex. I say this with confidence in the world-wide work of the many expert scientists interested in our problem, who are the products of modern physico-chemical training and skilled in the use and understanding of the many, new, and complex scientific tools at hand.

There are a number of firmly established, well-equipped and supported laboratories for ophthalmic research in various parts of the world, a few new ones since World War II, in which these men can do continuous and uninterrupted work on the many unsolved ophthalmic problems of which glaucoma is, as my dear colleague Sourdille says so aptly, "public enemy number one." There are still not enough of these workshops and not nearly enough of the qualified men, but we have made a splendid beginning.

In accepting this assignment, I was of course aware of the great difficulty of doing justice to the subject and the necessity of keeping an open and impartial mind on the many controversial points that are found therein. It was not, however, until I began to dig into my work that an overwhelming sense of frustration began to manifest itself.

This sense of frustration, although still present, as you can easily judge for yourself, has been considerably lessened and my task made more easy, fortunately, by the recent appearance of two fine books on this subject—Sugar, *The Glaucomas*, and Weinstein and Foldes, *Glaucoma: Pathology and Therapy*—as well as the timely (for me) appearance of annual reviews on glaucoma by Scheie and Haas in the *Archives of Ophthalmology*, and by Goldmann's most thorough review in Streiff's *Advances in Ophthalmology* published in 1952.

I have borrowed heavily from their material and am grateful to the authors for their work, in saving me the seemingly hopeless task of reading and evaluating the

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thousands of articles in all languages on glaucoma that have appeared even since 1830. This is a veritable Tower of Babel made of paper reaching far up in the sky and with, as usual, the most rarefied atmosphere at the top.

Brief history

The history of glaucoma, from the times of the Greek authors, Homer, Hippocrates, Plato, Aristotle, and Galenus (who gave us the word "glaukos") to the present, shows a steadily decreasing confusion in the understanding of this word as a diagnostic term of an ocular disease.

We are told that it first meant "glossy" or "blue," later in the early 19th century "green cataract," by which glaucoma came to mean a complicated cataract with complete amaurosis. Hardness of the eyeball was not an essential part of the diagnosis until it was first established by Richard Banister in 1622. Sir William Lawrence (1829) apparently was the first to use the term "acute glaucoma" but did not recognize the increased ocular tension as the cause.

A year later William MacKenzie emphasized the hardness of the eye and later (1844) recommended a puncture of the vitreous to soften it, although others had preceded him in describing the relatively good result of a limbic puncture in certain hitherto unrecognized conditions. However, in the fourth revised edition (1855) of his *Treatise on the Diseases of the Eye* this "great and astute Scottish clinician," as Duke-Elder calls him, described most eloquently an attack of acute glaucoma which he was still calling "acute choroiditis."

It was not, however, until the invention of the ophthalmoscope by von Helmholtz (1851) that a means of studying and recognizing glaucoma was evolved. Weber (1855) was the first to demonstrate cupping of the disc and three years later Müller discovered the histologic appearance of an excavated disc and ascribed it to the increased intraocular pressure.

To the immortal master, Albrecht von Graefe (1856), however, must go the major share of credit, for he explained all of the symptoms of glaucoma as the result of increased intraocular pressure and directed his genius to the problem of lowering it, with what great success all of us here are gratefully aware.

In the *Graefe-Saemisch Handbuch der gesammten Augenheilkunde* (1877), there are 138 pages devoted to the subject "Glaucoma" by Prof. Herman Schmidt of Marburg. This is an extraordinarily modern exposition and should make us sad to think that in these last 75 years we have not made greater progress. The opening sentences of Schmidt's article, which we may assume to represent the philosophy of glaucoma up to that time, read as follows:

"In the multiform picture of glaucoma three principal tendencies can be recognized as pathognomonic (a) the increase in intraocular pressure (recognized by the increased hardness of the globe), (b) the consecutive excavation of the optic disc, and (c) the inescapable blindness without therapeutic intervention.

"The course of the disease may be without or with inflammatory symptoms. In the former case, we have the simple glaucoma; in the latter, the inflammatory glaucoma: here again, we differentiate according to appearance and cause, an acute, a chronic, and an intermittent form. However, the various forms frequently merge. For instance, a simple or chronic glaucoma suddenly may show the picture of an acute attack and, on the other hand an acute glaucoma not infrequently changes into a chronic. Besides these genuine forms, these glaucomatous processes should be mentioned which occur in other eye affections. They are called secondary glaucomas."

Then Schmidt goes on to describe the symptoms and signs of inflammatory and noninflammatory glaucoma in such a fashion that, with few changes, his text could be considered standard for today. For instance,

in acute inflammatory glaucoma, the characteristic attack with pain, halos, shock, edema, and insensitivity of the cornea, the congested anterior segment, the narrow chamber, the semidilated and vertical pupil, rapid loss of sight, and the hardness of the eye are classically described.

The chronic form called simple, with its absence of pain and congestion, gradual loss of sight involving first the nasal field; the ophthalmoscopic findings with the "glaucomatous halo around the disc," the nasal displacement of the central vessels, the gradually increasing cupping, and the presence of arterial pulsation can also be considered as a classical picture.

Furthermore, the incidence, age and sex, racial factors, psychogenic factors, rise of tension in the dark, the influence of mydriatics precipitating an acute attack, tonometry, and many other factors are described in phrases that have a decidedly modern tone.

The still puzzling condition known as pseudoglaucoma or low tension glaucoma—or as v. Graefe called "amaurosis with excavation of the optic nerve" (he regarded it as a genuine disease of the optic nerve)—created as much controversy then as it does now.

In discussing the mechanism of the excavation of the disc, Schmidt says:

"The glaucomatous excavation as first emphasized by Heinrich Müller is the result of an increase in the intraocular pressure. Because of it the lamina cribrosa, as the weakest and thinnest part of the scleral coat, is retrodisplaced and excavated. However, this result is not due to a single brief, though high increase in pressure as in an acute attack of glaucoma, but the result of a longer existent pressure. Naturally the higher or lower capacity of the lamina cribrosa to resist has to be taken into consideration. At times it may be able to resist an increase in pressure for a long time, and other times it yields to a relatively low pressure which hardly exceeds physiologic limits. The latter is frequently seen in simple glaucoma."

Schmidt is no more satisfied with the explanation of the cause of the nasal contraction of the field of vision, solely on the basis of the excavation of the disc, than we are. He believed that a faulty blood supply of the retinal periphery should be considered, and said, "It should be mentioned that some cases with a deep glaucomatous excavation, but a relatively good vision, can be observed."

I regret that time does not permit me to give you the whole of Schmidt's article, for it is most revealing. It deserves republication and translation in its entirety.

But what interests us chiefly today is his discussion of the etiology of glaucoma. Here we find exposed a beautiful discussion of the neurovascular theories which have an extraordinarily modern ring. After many pages of discussion of the experimental and clinical studies of investigators whose names are so well known to us today, Schmidt's summary is as follows:

"After all these considerations we come to the following results. The greatest influence on the increase of the intraocular pressure and the consecutive appearance of a glaucomatous process is created by (1) the rigidity and loss of elasticity of the sclera, (2) conditions of irritation in the area of the trigeminal nerve (considered to be the vasodilator nerve of the eye), and (3) congestions in the venous bed of the eye. The sympathetic is only of minor importance."

In 1830, Friederich Schlemm reported his discovery of the circular canal at the border of the sclera and cornea. However, the significance of this structure was not realized until 1869 when Schwalbe, by his famous injection experiments, showed that Prussian blue injected into the anterior chamber, in time reached the anterior ciliary veins. This led to the famous controversy with Leber beginning in 1871, so fruitful for us in the end, by stimulating other famous investigators to determine the exact nature of the outflow channels.

Although in 1876 the basic information regarding the outflow of aqueous already was

known, Schmidt, for some reason, chose to belittle it as a factor in glaucoma. He says that a pathologic increase in intraocular pressure can be explained "only in two ways: either there is an abnormally large content or there is an abnormally small width and elasticity of the eye compared with the content. There are two causes for the abnormally large content: either there is an increased addition of fluid above the physiologic or not enough drainage." However, it was not until about 1880 and up to the turn of the century that the venous nature of Schlemm's canal and its major function as an outflow channel of aqueous was finally accepted.

In 1876, three years after Leber had demonstrated the escape of the aqueous fluids at the angle of the anterior chamber, Max Knies and Adolph Weber independently showed that, in eyes blinded by glaucoma, this outlet is commonly obstructed. A peripheral adhesion of the iris in such eyes had been incidentally noted by previous writers, but its significance was now recognized for the first time.

The retention theory of glaucoma got off to a slow start, but after the work of Priestley Smith particularly, and many others before and after, this theory has not only held its own for the last 50 years, but most recently has enjoyed renewed popularity.

In 1898, Priestley Smith said, "It is perhaps not surprising that the so-called retention theory of glaucoma is not yet accepted by all ophthalmic surgeons, for the most acute observer, studying the matter only from the clinical point of view, sees nothing of the normal filtration process and little of the special conditions which disturb it in glaucoma; but for those who have investigated the subject thoroughly in the laboratory as well as in the consulting-room, the evidence is complete that retention is the essential factor in the morbid process."

However, I am afraid that I have already devoted too much time to the historic background of our subject. Enough has been said to show that for the last 75 years the workers in this vineyard have considered the chief factors in the etiology of glaucoma as being:

a. An increase in the intraocular volume, whether due to increased "secretion of aqueous" or not.

b. A retention of the intraocular fluid due to obstruction in the outflow channels of the eye.

The increased volume has for all these years been considered as controlled by vasomotor or neurovascular factors, and the obstruction of the outflow, until more recently, was thought to be either the result of the first, or at least of relatively minor importance. So we see that the controversy between the so-called mechanical school and the neurovascular school is indeed a very old one, and is still not resolved. It is the purpose of this paper to continue this controversy in the light of recent discoveries affecting both sides of the question.

Definition of primary glaucoma

Professor Schmidt's definition, which holds good today, has already been mentioned. There are, however, many other definitions; such as the one given by Priestly Smith (1879): "Glaucoma is, then, strictly speaking, a symptom—a complex one, truly, but still a symptom—of certain hidden processes which lead to excess of pressure within the eye.

"Those forms of the disease in which increase of tension is the first discoverable change are termed primary, whilst those in which it has been preceded by other evident morbid processes are classed together as secondary, or consecutive."

Jonas Friedenwald writes: "Glaucoma is not a disease but a whole complex of diseases which have as their common feature an abnormal elevation of intraocular pressure." And he introduced the term "normative" as being more precise in meaning, being that intraocular pressure which is compatible with continued health and function.

Kronfeld has defined glaucoma as, "The ocular diseases comprised under the term

the glaucomas are characterized by a progressive loss of visual function which runs parallel to and is in all probability directly caused by a state of elevated intraocular pressure."

Duke-Elder says, "The term glaucoma does not connote a disease entity, but embraces a composite congeries of pathologic conditions which have the common feature that their clinical manifestations are to a greater or less extent dominated by an increase in the intraocular pressure and its consequences."

We see, therefore, some discrepancy in the definition of glaucoma, low-tension glaucoma, and of pseudoglaucoma. However, we all know what we mean by the term pseudoglaucoma which has been used for almost 100 years. And certainly since Professor Schmidt's time the primary glaucomas have been divided into two groups—the acute (congestive) glaucoma and simple (noncongestive) glaucoma.

One would think that, with the clear understanding that all of us have regarding these two types, there would be no room for further confusion. However, this is not the case. Modern textbooks in ophthalmology continue to group the two forms into a symptom complex, in spite of the fact that it can be shown, particularly by means of modern technical methods of examination, that the two are quite different.

There is, indeed, a growing belief particularly in the United States that the acute form of primary glaucoma, the so-called congestive type, is a definite entity based exclusively upon a narrow angle of the anterior chamber. For this reason the workers in the United States have adopted the classification of primary narrow-angle glaucoma for the congestive form; and wide or open-angle glaucoma for the noncongestive or simplex type, since in the latter condition there is no evidence that blockage of the angle of the anterior chamber, at least in the early stages of this disease, plays any role.

The term wide angle does not mean that the anterior chamber is always a deep one. As a matter of fact the anterior chamber may be as shallow as that seen in the first type (narrow angle) but, based on gonioscopic studies, the angle is open and there appears to be no obstruction that prevents the access of aqueous to the canal of Schlemm.

Many of us believe that this classification has helped us to a better understanding of the problem. Advocates of this classification have been placed in the category known as the "mechanical school." In contrast there are those who believe that the primary glaucomas are of neurovascular origin. This controversy is producing much fruit in our present investigations.

Now one would also think that having been established that there are two forms of glaucoma, as above defined, there would be little argument as to classification of the types, both primary and secondary. But this is not true, since we find that there are at least eight different classifications by v. Graefe, de Grosz, Elschnig, Schmaudigel, Raeder, Rosengren, Barkan, and Sugar. There are probably others of which I am unaware.

However, the conventional classification of primary glaucoma which is familiar to most of us throughout the world is:

1. Chronic simple (noncongestive, compensated).
2. Acute congestive (incompensated).
3. Chronic congestive (incompensated).
4. Absolute.

Many believe that these forms are different types of the same underlying process, and that one can have in the chronic or simple type of glaucoma superimposed the acute or congestive type. Furthermore, in the chronic simple form the anterior chamber can be quite narrow producing or giving rise to the narrow chamber angle type of glaucoma as a result of a number of conditions including surgical intervention which produces at times embarrassing edema of the ciliary body.

It is also confusing to recall that each sub-

sequent attack of acute glaucoma in the narrow-angle type will produce more and more synthechias and thus convert the narrow-angle type of glaucoma into the chronic simple form, with or without congestion. Unless corrected, all types end in absolute glaucoma and blindness.

Finally, to add to the confusion, there is a subtype of primary glaucoma of the simple form, rare in my experience, where the ophthalmoscopic picture is characteristic of the glaucomatous disc and yet tonometric measurements show a normative range of tension. And in this so-called low-tension glaucoma, which is a true condition, carefully repeated tonometry will reveal tension readings of the diurnal variation type that cannot be distinguished from the high-tension group. Furthermore, tonography here reveals a decreased facility of outflow of aqueous.

In another group of cases where the signs of simple glaucoma are quite evident and yet the tonometer readings show little or no elevation above the normal, scleral rigidity is implicated, and this seems to be particularly true in the myopic eye.

On the other hand, there are cases of pseudoglaucoma where the ocular tension and tonography is always normal and yet the nervehead and fields of vision are characteristic of glaucoma. The cause of this condition is nutritional. True low-tension glaucoma should, however, be sharply differentiated from the cases of pseudoglaucoma in which a cavernous atrophy (Schnabel) of the optic disc or sclerosis of the nutrient vessels of the optic nerve (Cristini) or pressure on the optic nerve by a calcified and dilated internal carotid artery (Knapp, Theil) may be responsible.

Not infrequently we find that our patients with simple glaucoma, in whom the intraocular pressure has been maintained at a normal level or below by surigical or medical means, continue to show further cupping, excavation, and atrophy of the optic nerve. Associated with this is further loss of fields of vision and finally total loss of sight. This conversion of a true glaucoma into pseudoglaucoma is explained by (a) marked sclerosis of the nutrient vessels, (b) a rapid yielding of an already architecturally weakened lamina cribrosa even to low intraocular pressure, or (c) both.

Physiology of inflow and outflow of aqueous

It seems necessary here to discuss briefly the current beliefs regarding the formation of aqueous and its passage through and out of the eye, for in its simplest terms intraocular pressure is a result of the balance between aqueous inflow and aqueous outflow.

The blood-aqueous barrier, according to Duke-Elder, is composite and varies in different parts of the eye, but the common factor of importance must be the capillary walls.

In the posterior part of the eye, the effective barrier is formed by the retinal capillary walls. In the central region, the barrier is composed of the walls of the uveal capillaries plus the anterior portion of the retina and its prolongation, the ciliary epithelium and the epithelium on the posterior surface of the iris. Most anteriorly on the anterior surface of the iris, the barrier consists essentially of the naked uveal capillaries alone.

Intraocular fluid is diffused through the endothelial cells of capillary walls and presumably not through the intercellular spaces. In addition to the diffusion factor, secretion plays a part. There is no place here to discuss the evidence for these opinions, although a knowledge of the formation of the aqueous is an essential part of the understanding of the glaucoma problem, since an increase in its formation will lead to an increase in volume in the eye, and perhaps an elevation of intraocular pressure, or a decrease in its formation results in softening and shrinkage of the eyeball.

In spite of the enormous amount of work devoted to the problem of the formation and intraocular circulation of the aqueous, many questions remain unanswered. There is no

adequate theory of the formation of aqueous humor that satisfies all these questions. A recent paper by Davson reviews this subject and those who are interested are urged to study his paper carefully.

I think that all we need to say here is that aqueous is formed continuously as a clear almost protein-free fluid by the ciliary body ejecting into the posterior chamber and from thence into the anterior chamber where it leaves the interior of the eye through the canal of Schlemm system. It presumably does this by percolating through the meshwork of the sclera, passing through the endothelium lined canal, from here into the capillaries that empty into the vessels of the intrascleral venous plexus.

As a result of the work by Kiss (1943), it is believed that the aqueous not only goes through the canal of Schlemm but discharges into the ciliary plexus adjacent to and within the region of the ciliary mechanism, from thin veins branching off to the episclera. As evidence of this one occasionally sees the emissarium cysticum.

The discovery of the aqueous veins in 1941 by Ascher has stimulated an enormous amount of interest and work. There is no question now that these structures play an important role in the drainage system. They arise in the canal and connect with the conjunctival vessels as Ashton has so beautifully shown.

It is still uncertain exactly what role, if any, the anterior iris endothelium plays in the absorption of the aqueous. The question of whether the anterior surface of the iris actually is covered by this endothelium membrane is still unresolved.

Davson believes that the diffusion from the posterior surface and also from the anterior surface of the iris plays a part in the formation of the aqueous. This is a controversial point and needs further study. So far as the absorption of material from the aqueous the question still remains, for example, of how fragmented blood cells in the aqueous are absorbed, if they are, by vessels in the iris, for according to Kiss the plexus ciliaris proved to be a most important resorption region.

Returning to the secretory activity of the ciliary processes (and doubtfully the iris) it is well known that the secretory activity can be influenced by osmotic changes. An increase in the permeability of the capillaries will permit a formation of a filtrate leading to high protein content which in turn influences the osmotic pressure.

Hypotonicity of the osmotic pressure of the blood may reflect itself in an increase of the intraocular pressure and vice versa.

The works of Friedenwald and of Kinsey may be of great significance in this particular point. They have shown that more water diffuses into the posterior chamber than diffuses out again. The excess of water into the posterior chamber dilutes other substances. The aqueous humor thus formed flows between the iris and lens into the anterior chamber where there is a further exchange of nonelectrolytes. The electrolytes do not exchange because of the walls of blood vessels of the iris being impermeable to ions under normal conditions. All constituents of the aqueous humor of the anterior chamber then escape by flow from the eye at the angle at a rate of about three microliters per minute.

The influx of water as a result of hydrostatic and osmotic pressures is responsible for intraocular pressure. The magnitude of the pressure depends on the rate of production of hydroxyl ions in the epithelium of the ciliary body and, to a less extent, the porosity of the blood aqueous barriers. Under normal conditions these factors are in balance.

The hydroxyl ions by reaction with carbon dioxide are converted to bicarbonate ions. These are electrically neutralized by sodium and other anions diffused from the blood. Kinsey has also shown that the concentration of bicarbonates and ascorbates are higher in the posterior aqueous than in the anterior aqueous. Thus here note that a very slight change in the osmotic pressure within

the eye is capable of producing an enormous change in the intraocular pressure.

It is on the basis of this information that Becker has recently shown that a carbonic anhydrase inhibitor, Diamox®, offers an exciting new tool for the investigation of the problems of glaucoma and its discovery is of the utmost significance. Diamox® (2-acetylamine-1,3,4-thiadiazole-5-Sulfonamide), which can be administered with safety to humans orally in doses of 500 to 1,000 mg., is an effective agent in lowering intraocular pressure. Although its mode of action is not as yet completely determined, there is no doubt that the formation of the aqueous is more or less inhibited by the use of Diamox®.

If dilatation of the arterioles and capillaries occur, the change in the hydrostatic pressure will depend on whichever of the two influences predominates. If an obstruction occurs in the venous stream, back pressure becomes effective in the capillaries and a rise in intraocular pressure results.

An increase in the volume of blood through vasodilatation will lead to an increase in intraocular pressure due to the indistensibility of the sclera, provided that the efficiency of the drainage channels is embarrassed.

Finally, inefficiency of the drainage channels will lead to an increase in pressure either through the organic constriction of the exits or by a rise in the pressure in the veins outside the sclera to a point above the level of the intraocular pressure.

One can recapitulate by saying that aqueous is continuously formed probably by the anterior uvea, particularly through the ciliary processes and continuously draining into the anterior scleral vessels through the drainage channels.

When we consider the part that the exit of aqueous from the eye plays in the control of the intraocular pressure we are on firmer ground. A recent paper by Becker and Friedenwald splendidly summarizes for us our present knowledge of clinical aqueous outflow. These authors point out that, although it has been known for many years that patients with chronic simple glaucoma had decreased facility (increased resistance) of aqueous outflow, it is the work of Goldmann and Grant that has placed it on a quantitative basis.

I should like to pause here to pay tribute to the late Mark J. Schoenberg who said in 1912, "The degree of intraocular pressure depends in a great measure on the integrity of the drainage system of the eye and the examination of the rate of ocular drainage is of more importance than the simple measurement of the intraocular pressure. The rate of ocular drainage can be measured by aid of the Schiøtz tonometer in animal and human eyes with a certain amount of accuracy."

Schoenberg was among the first, I believe, to apply the Schiøtz tonometer to the eye for three minutes or so and to note the change in the readings of the ocular tension during this time, considering it as a measure of the rate of outflow of aqueous.

Goldmann, in 1949, described a fluorometric method of measuring the minute volume of the anterior chamber. It is 2.2 cu. mm./min. +0.37. The pressure under which the intraocular fluid drains into the episcleral veins he called the outflow pressure.

A number of other workers, notably Löhlein and Weigelin; Linnér, Rickenback and Werner; Grant, et al., are in good agreement that the mean value of the normal pressure in the aqueous veins is 9.7 mm. and that of the outflow pressure is 7.2 mm. Thus the resistance of this system is 3.2 calculated according more or less to Poiseuille's law of hydrodynamics.

Grant, using the tonographic method that he developed, found the value of the resistance to aqueous outflow in the normal eye to be 5.0 mm. He thus obtained a minute volume of 2.4 cu. mm. which is in surprisingly good agreement with the values of Goldmann. These workers, using entirely different methods of investigation, have likewise obtained comparable values for the

average normal coefficient of facility of outflow (Grant 0.22, Goldmann 0.33).

The factors that determine the rate and ease of outflow, according to Duke-Elder, are:

1. The number of efferents from the canal of Schlemm (approximately 30).

2. The pressure difference between the canal of Schlemm (20 mm. Hg) and the episcleral plexus (10 mm. Hg roughly).

3. The nature of the outlet vessels (aqueous veins). There is considerable variability in the radius, length, straightness, branching, and the nature of the inlet and outlet of aqueous veins.

4. The viscosity of the aqueous humor (in the normal case about 1.1 compared with water = 1).

5. The temperature to which the system is exposed (about 10°C. between the intraocular temperature and that in the subconjunctival tissue).

Any alteration therefore in any or all these factors will affect the rate and ease of flow in the drainage mechanism. No doubt there are other factors, as yet unknown, which play a part in controlling the opening and closing of the sluices that drain the eye as the necessity for normal intraocular pressure may require. We have gone a long way, however, in having a better understanding of these forces in the last few years.

Before leaving this phase of the subject it is necessary to emphasize that the two normal eyes of an individual appear to act synchronously insofar as the pressure is concerned. This is particularly true of the phasic variation of pressure and the volume of normal aqueous outflow (Grant 2.4 cu. mm. per min., Goldmann 2.2 cu. mm. per min.).

Physiologic control of intraocular pressure

Our understanding of the physiologic control of intraocular pressure is for the most part speculative and controversial. Studies on the basic and fundamental problem continue apace and seem to be pointing the way for us. One of the most important landmarks, I think, that we have already passed is the discovery by Maslenikow (1904) of the diurnal changes of intraocular pressure.

Since then, this physiologic phenomenon has been intensively investigated, particularly in the last few years and especially by the workers at the Institute of Ophthalmology in London, of which Duke-Elder is the articulate spokesman. I am sure that I cannot improve on his description of the regular and rhythmic variations of tension which occur in the normal and glaucomatous eye. Hence this direct quotation:

"The normal diurnal variation in intraocular pressure, which never exceeds 5.0 mm. Hg, is a well-known phenomenon which has not received sufficient attention. It would seem that in this respect each individual has a characteristic rhythm which is obstinately maintained in spite of all environmental variations, and that both eyes vary together. It is important that the curve is unaltered by bodily posture or activity, and is unaffected even if the patient's habits are suddenly reversed and he remains up at night and spends the day in bed. Osmotic changes are not at fault for the rhythm is unaffected by rest or work, by changing the times or the richness of meals, or by feasting or fasting. Nor is the size of the pupil, which might hinder the flow of the aqueous humor at the angle of the anterior chamber, an effective factor, for it has been found that the rhythm occurs whether the pupil is fixed in dilatation or contraction by atropine or pilocarpine, if the continuity of the iris is broken by an iridectomy or a coloboma, or even if this tissue is congenitally absent in aniridia. Any action of light upon the ocular capillaries is negatived by the same monotonous persistence of the rhythm if a brilliant light is maintained all night and the day is spent in darkness. It has been suggested that message of the eye by muscular movements through the day aided the circulation of the intraocular fluid and lowered the pressure of the eye, while the relative immobility during sleep has the opposite hypertensive effect owing to stag-

nation of the aqueous humor; but again, the maintenance of the characteristic rhythm of the pressure despite the reversal of habits has disproved this.

"The only circumstance in fact which has been shown to alter the incidence of these oscillations is a long-term and fundamental change of habits, for if a complete reversal of the habits of work and sleep are established over some time, a similar reversal of the variation in ocular tension occurs. It would, indeed, seem most probable that the basis of these diurnal changes is associated with the rhythmic variations which so commonly occur both in vegetable and in animal life—affecting, for example, sleep, the temperature, diuresis, the electrolytic content of the blood, and other basic functions. It would seem that each individual has a characteristic rhythm which is obstinately maintained, and that both eyes habitually vary together, circumstances which make is likely that the periodic alteration is determined by a habitual hypothalmic rhythm imposed upon the organism by long-standing environmental conditions. The fact that such rhythms as the diurnal variation in temperature are apparent in the newborn infant indicates that these fluctuations are very fundamental and probably innate.

"However that may be, it would seem that a regulating mechanism must exist which tends to maintain the intraocular pressure at a physiologic level, within a slight habitual rhythmic variation, in spite of the drastic interference which operative procedures may entail. In early cases of glaucoma the first alteration in tension is not so much a rise as a distortion and exaggeration of this normal diurnal variation. It is as if the glaucomatous eye has lost some power of vascular control so that, instead of the normal slight variation of tension, a gross and uncompensated swing occurs."

Many workers believe that general arterial pressure plays a minor role in the control of intraocular pressure in the normal eye. On the other hand, there is considerable scientific, clinical, and experimental evidence that venous pressure in the posterior segment of the globe, particularly the vortex veins and in the anterior portion, chiefly the episcleral venous network is a most significant factor. There is impressive evidence at hand to support the idea that the phasic diurnal variations of the ocular tension are closely associated with the same sort of variation in the venous pressure and definitely not in the arterial pressure.

However, local arterial pressure may be of importance in the control of ocular tension. For example, a considerable capillary engorgement may produce a spectacular rise in intraocular pressure, attributable primarily to an exudation, normally held in check by the physiologic tone of the vessels—of a protein-containing fluid into the anterior chamber from the iris. Moreover, the increased volume of blood within the globe under these conditions must be a factor in the raising of the intraocular pressure.

Arkin thought that a failure of constrictive ability in the ciliary arteries was responsible for this vasodilation. About 40 years ago Van Lint wrote that he considered turgescence occurring in the choroid or uvea from any reason whatsoever as leading to a veritable erection of the ocular globe. Duke-Elder has recently expressed the same idea. The cause of this physiologic phenomenon is debatable.

Experimental and clinical evidence by many astute observers strongly supports the belief that vasomotor activity is responsible. Just how this is controlled is still unknown. We label it "neurovascular" and that is about as far as we can go with complete certainty.

A recent paper by Weinstein (cortico-visual problems in glaucoma) discusses this problem very well and summarizes for us the various theories. The control tonus is certainly a complex function which is analogous to other vegetative functions and is dependent upon the action of numerous

(theoretic so far) centers in the cortex, diencephalon (hypothalmus), bulb or medulla, or even peripheral, that is in the sympathetic, the carotid sinus, or the parasympathetic ganglia. Indeed, if you study the literature thoroughly you will find that there scarcely remains a part of the central nervous system or contents of the orbit in which this hypothetical center of control of ocular tension has not been placed. Likewise hormonal influences (for example, the pituitary) have been blamed.

Weinstein also draws attention to what are called interoceptors, particularly baroceptors. These seem to be a special apparatus of nervous tissue contained in all internal organs. Their function is to report the state of tension within that organ, by afferent pathways through the spinal cord.

The intercalary cells reportedly found in the iris by Lavrentjev, Boeke, and Feyrter are assumed to be the histologic interoceptor that signals changes in the intraocular pressure to the proper authority for suitable control. All of this makes fascinating reading, but I find it impossible to come up with any clarification of my state of utter confusion in this matter, and shall leave to the future and to those more skilled to settle the matter.

Simple (open-angle) glaucoma

This form of primary glaucoma is characterized by an insidious onset and a slowly progressive and ruthless destruction of vision. It is associated most often with increased intraocular pressure that tends to become higher as the disease advances. In contradistinction to the acute inflammatory type, the angle of the anterior chamber is open as seen gonioscopically although the chamber itself may be deep or narrow. It is a disease of age and is more common in the male.

Duke-Elder and his group have intensively studied the rhythmic wild oscillations of phasic variations of the intraocular pressure in this condition. It is as if the normal phasic variations are out of control for some reason unknown. They found that there are three groups:

Group I (20 percent of the cases) shows a falling type of curve. The tension is high on waking in the morning, reaching a peak thereafter and gradually falls, with some fluctuations, until midnight.

Group II (25 percent of the cases) shows a rising type of curve. The tension is low in the morning, climbs steadily throughout the day until about between 4:00 and 6:00 p.m. and then falls steadily throughout the evening and night.

Group III is most common (55 percent of the cases). It shows a double variation, a sort of combination of Groups I and II. The tension rises in the early morning, reaches its peak in the forenoon, and falls to reach a minimum in the early afternoon, followed by a secondary rise at about 6:00 p.m., and then declines steadily throughout the evening and night.

It is generally agreed that this phenomenon of wild oscillations in ocular tension is of fundamental importance and of greater significance than is the height of the tension, especially in early diagnosis. Furthermore, as the disease progresses the phasic swing becomes smaller and the base pressure and peak pressure tend to merge, although there are exceptions to this rule. The pattern of the oscillations in the two eyes of the same individual is fundamentally the same.

Duke-Elder points out that the rhythm is characteristic of the individual, is independent of blood pressure, age, sex, refraction condition, and width of angle of the anterior chamber, and is seen in the earliest stages of the disease, often before glaucoma is clinically evident. The same individuality is preserved after a successful drainage operation, although the excursions are lessened. Only underlying circulatory instability can explain such behavior.

Pau measured the ocular tension of 10

children with buphthalmos (hydrophthalmos) and found that the phasic variations in these cases were similar to those in primary glaucoma of the adult. If this is found to be generally true, it seems to be an argument in favor of the "mechanical" concept. In this condition there is structural mechanical blockage to the outflow of aqueous and presumably no sclerosis of the capillaries as seen in the aged.

While it is true that there is slim possibility that, in these cases of hydrophthalmos, the "structural sclerotic impediment to drainage can be effective at 10 o'clock and ineffective at 12 o'clock in the same day" (Duke-Elder), the increasing or decreasing rate of formation of the aqueous could well be the chief factor. Against this is the cogent argument that there is good evidence that increased capillary permeability and increased diffusion of aqueous do not play a part in the causation of the periodic rises of tension.

Duke-Elder puts this neatly when he says that the pressure changes are due to one of three things.

a. A variation in the capillary blood pressure. There is good evidence that changes in the vascular pressure and particularly in the capillary pressure will have almost immediate effect upon the exit veins, because of the direct connection between uveal arteries and veins.

b. A variation in the volume of the intraocular fluids. All the evidence so far accumulated points to the conclusion that there is, at least, no alteration in the normal standard permeability curves nor in diffusion of aqueous, except during the attack of an acute inflammatory glaucoma. While most of us, at the moment, believe that there is no evidence of an increased volume of intraocular fluid in primary simple glaucoma, new evidence, based on the study of the effect of Diamox®, may change our opinion.

c. A variation in the drainage of the intraocular fluid. During the increasing phase of tension there is little or no flow through the aqueous veins, during the stage of level tension the flow is average and during the stage of decreasing phase the flow is accelerated. When pressure is applied to a laminated aqueous vein, blood influx is seen to occur during the ascending phase of pressure and aqueous influx during the descending phase. In other words, the venous pressure is higher than the aqueous in the ascending phase, and lower in the descending phase.

When the sympathetic and parasympathetic nerve supply to the eye is abolished by anesthetizing the ciliary ganglion, it not only abolishes the entire phasic variation of pressure on the same side, but has a dampening effect on the phasic variation on the opposite side. Furthermore, if the stellate ganglion (sympathetic) is blocked, the phasic variation is similarly abolished.

Sympatholytic drugs (adrenolytic such as hydralazine hydrochloride, pentolamine hydrochloride (Regitine, Ciba), and piperoxan hydrochloride, and agents that have both a parasympatholytic and sympatholytic action such as hexamethonium bromide or tetraethylammonium chloride (etamon chloride, Parke Davis) also abolish the phasic variations of intraocular pressure.

Duke-Elder also points out that cholinergic drugs, such as pilocarpine and eserine, have hypotensive effect, not so much as the result of pupillary constriction but particularly as the result of their dilating effect on the capillaries and in addition in the increase of the permeability of the capillary walls, as seen with the fluroescein test.

The effect of these drugs on the aqueous veins has been readily observed. After the instillation of philocarpine, for example, aqueous influx into an afferent blood vein is rapidly accomplished, even when this phenomenon could not be elicited on pressure over the vein before the instillation of pilocarpine.

It is concluded that irritability, instability, and constriction of the capillary circulation—so-called sympatheticotonia—produces a rising tension in glaucomatous eyes. The

tension falls when the circulation recovers its normal equilibrium.

This is manifested when there is a capillary dilatation dissipating blood over a wide bed, lowering its hydrostatic pressure, and allowing the pressure in the exit veins to fall.

This is accomplished, then, not only when the ciliary and stellate ganglions are blocked or when methonium compounds and miotics are used, but also with the physiologic vasodilation that occurs after rest, sleep, and the application of Dionin and heat.

It is also speculated that vasodilation in the descending phase may be the result of the response to increased intraocular pressure itself, anoxia, the formation of histamine, and the opening up of accessory drainage channels.

But these at the moment are theories only. However, in spite of this, the evidence for the neurovascular background of chronic simple glaucoma is indeed impressive and many competent and thoughtful observers are enthusiastic in their championship of this idea.

On the other hand, tonography and bulbar pressure tests (Blaxter) have unquestionably demonstrated that there is an abnormal resistance to aqueous outflow in chronic simple glaucoma. Furthermore, in a number of cases of glaucoma simplex in which Diamox® has been given, there results a fall in the intraocular pressure down to a certain point, still abnormally high, beyond which it will not go without the instillation of miotics.

Tonography reveals in these cases that there remains the same degree of resistance to outflow of aqueous that was present before the use of the drug. This is a sort of "law of diminishing returns," and is particularly significant when resistance to outflow is more pronounced when greater demands are made upon the outflow channels.

The site of the resistance to aqueous outflow is still uncertain. There are those who firmly believe that it lies in the trabeculum. Here there is primary sclerosis and thickening of the trabeculum. Others have found a deposition of pigment and "scars" in the trabecular spaces.

Herbert and Fortin each believe that there is a defective pull of the ciliary muscle against the scleral spur so that the trabecular spaces and Schlemm's canal cannot be properly opened.

Friedenwald believes that there was a failure of the osmotic pressure effect on the aqueous in Schlemm's canal due to failure of the sclerosed afferent arterioles of Schlemm's canal to bring in sufficient plasma. However, Ashton has recently shown that there apparently are no afferent vessels in Schlemm's canal.

Kronfeld's studies, on the other hand, give some support to Friedenwald's hypothesis. He found that in glaucoma simplex the sudden lowering of intraocular pressure results in an inadequate filling of Schlemm's canal with blood, significantly differentiating these cases from normal eyes. It is obvious that many microscopic studies of eyes affected with simple glaucoma, particularly in the early stages, must be made before we know the answer.

There is much in the literature regarding the role of vascular sclerosis in the picture of glaucoma, particularly the constriction and sclerosis of the arterioles of the uveal tract and the nutrient vessels of the retina and optic nerve. This has already been discussed briefly. There is considerable evidence that sclerosis of the intraocular vessels plays a large part in the overall picture of simple glaucoma, but whether this role is a causal one, or a part of the aging process in general, or hastened by the increased intraocular pressure effect is still not clear.

Acute inflammatory (congestive or narrow-angle) glaucoma

There is an increasing number of investigators who believe that acute inflammatory glaucoma is an entity that is entirely distinct and separate from simple glaucoma. The arguments in support of this claim, based on

experimental and clinical studies made by many competent observers, are strong and convincing. I believe that if this concept is accepted it will clarify our problem and I plead here for its universal acceptance.

The inclusion of all forms of glaucoma under a unit category has led us into many difficulties, not only of understanding and interpretation, but particularly in regard to empirical forms of medical and surgical treatment.

In contradistinction to simple glaucoma, congestive glaucoma is more common in the female, more frequently hereditary, is diagnosed at an earlier stage, is frequently (some claim invariably) associated with a shallow anterior chamber and a narrow chamber angle (bilateral). A high percentage (80 percent) of these patients are hypermetropic.

The onset of congestive glaucoma is acute and not insidious, the attacks are periodic, characterized by complaints of blurring of vision, ocular pain, and seeing of halos around lights. The elevation of ocular tension is episodic, may be precipitated by emotional worry or strain, darkness, menstruation, fatigue, mydriasis by drugs, or on viewing motion pictures; it is relieved by sleep.

Cupping and atrophy of the disc is not found early in the course of the disease, nor do the changes in the fields of vision, diagnostically characteristic of the simplex form, occur except late in the evolution of the disease. The diurnal variation in intraocular pressure is normal except during the acute episodes. The fluorescein test shows normal concentration except during the acute attack when it remains high and the fluorescein disappears rapidly when the acute attack is over.

In cases of simple glaucoma the apparent outflow pressure remains abnormally high when the ocular tension is normalized, whereas in congestive glaucoma the apparent outflow pressure quickly becomes normalized. Or, to put it another way, during the acute stages of congestive glaucoma there is a marked obstruction to aqueous outflow which disappears when the attack is over.

Miller has shown that when the stellate ganglion is blocked by the injection of procaine:

a. In simple glaucoma there is produced an immediate rise in intraocular pressure followed by a fall.

b. In congestive glaucoma, when angles are open, there is an immediate fall in pressure, just as occurs in the nonglaucomatous eye.

c. If the angle is partially blocked, the pressure first rises and then falls as it does in simple glaucoma.

d. In cases where the angle is completely blocked, an acute congestive attack was produced.

It is interesting that he found that, after stellate ganglion procaine block in simple glaucoma, "the recipient laminated veins at first fill with blood and regain their laminated appearance when the intraocular pressure begins to fall." Whereas in "congestive glaucoma the aqueous veins remain clear throughout the period of observation if the angle is structurally open."

Miller concludes that obliteration of the stellate ganglion causes a sudden volume increase in the intraocular contents due to the dilatation of the intraocular capillary bed. Thus in an eye with congestive glaucoma there is a similar volume increase but, provided there are no pathologic changes in the angle, the outflow is assisted by the accompanying pupillary constriction and lowered venous pressure and the tension falls.

The changes produced by the injection of the stellate ganglion are of short duration except when the angle is completely blocked, and the procedure has no therapeutic value. But might it not be used as a provocative test?

Bangaerter, Goldmann, and many others have found by gonioscopy that the angle in congestive glaucoma was closed during the acute attack.

The shallow anterior chamber and narrow chamber angle in these cases, seen gonioscopically and even with the slitlamp, is an old

observation that is universal. It is, I believe, the key point of differentiation between the two forms of primary glaucoma. It accounts for the controversy between the "neurovascular" and the "mechanical" followers.

Essentially, this controversy lies between those who believe that obliteration of the angle is the result of the acute rise of intraocular pressure and those who believe that the rise of tension is due to the obliteration of the angle.

Actually, the truth probably lies between. There is no doubt about the vascular crisis that occurs and similarly there is no doubt about the pre-existence of a narrow angle in the majority of cases of congestive glaucoma. The combination produces a vicious circle.

In 1857, von Graefe, describing the symptomatology of glaucoma, observed that the anterior chamber often was shallow. Priestley Smith (1887) stated that the shallow chamber was a typical sign in acute congestive glaucoma. Raeder (1923) classified cases of glaucoma into those that had a shallow chamber and those that had chambers of normal depth.

Rosengren (1930-1931), using a method for measuring the anterior chamber devised by Lindstedt (1913) that was highly accurate, published a large series of measurements. His studies showed that the depth of the anterior chamber in glaucomatous eyes is, on the average, less than that in normal eyes and that the shallow chamber is not a consequence of the increase of intraocular pressure. He also concluded that "eyes which became subject to glaucoma as a consequence of a shallow anterior chamber are characterized by the occurrence of acute attacks in the majority of cases. In certain instances, however, the glaucoma takes the form of uncomplicated glaucoma simplex."

The gonioscopic investigations of Barkan, Sugar, Gradle, Kronfeld, Scheie, and other observers, chiefly in the United States of America, disclosed that the chamber angles in eyes subject to attacks of acute congestive glaucoma is noticeably narrow. Thus arose the term "narrow-angle glaucoma" as a substitute name for acute congestive glaucoma. Its counterpart "glaucoma simplex" was termed "wide-angle glaucoma." It is agreed that this is not a good term and many of us prefer the name "open-angle glaucoma" in its place.

In 1953, R. Törnquist published an important monograph on "Shallow anterior chamber in acute glaucoma: A clinical and genetic study." This monograph is worthy of the most careful attention.

Törnquist measured the chamber depth by a method devised by Stenström (1953) of a large series of normal eyes in various age groups. The sexes were evenly divided. He found that with increasing age a decrease appears from 3.19 mm. on an average at 20 years of age to 2.68 mm. at 65 years of age in males. The average values are somewhat lower in females.

An investigation was made of relatives of 49 patients with typical acute glaucoma with a shallow anterior chamber. He found that the chamber depth in the siblings as well as in the children is lower, on the average, than the normal values for the corresponding age. The differences (about 0.20 mm.) are statistically significant.

Furthermore, 45 pairs of twins with normal eyes were studied and the author concluded that the chamber depth is genetically determined.

Thus, as accumulative evidence continues to develop from clinical, surgical, and laboratory investigations, I find myself in complete agreement with the belief that acute congestive glaucoma occurs most frequently in eyes that have anatomically narrow angles associated with hypermetropia, which predispose them to attacks of glaucoma.

Acute attacks occur whenever the angle becomes blocked through any cause, proved or theoretical, such as increase in the volume of the vitreous, increase in the flow of aqueous producing a higher pressure in the posterior chamber, increase in the intra-

TABLE 1

PROVOCATIVE TESTS FOR GLAUCOMA

Test	Year of Report	Various Workers	Based On	Interpretation
USE OF ORAL, PARENTERAL, AND TOPICAL AGENTS				
Water-drinking	1928 1930	Schmidt Wegner	Studies of Marx on changes in blood by drinking water	Rise of over 9 mm. Hg to 33 or more, 6–15 mm. in half hr. in glaucomatous eyes—occurs at any phase
Adrenalin	1921 1947	Knapp Gurvich		1:1000 adrenalin hydrochloride 5 times at 1 min. intervals Glaucoma—pupil dilates
Caffeine	1925 1926	Wegner Lohlein	Intravenous injection of 0.2 gm. caffeine or 150 cc. water with 45 gm. coffee	Rise of up to 15 to 20 mm. Hg (Schiøtz) in 20 to 40 min.
Mydriasis	1926 1936 1948	Jackson Gradle Sugar	2% cocaine 2% euphthalmine 4% homatropine hydrobromide	No rise in tension in normal Increase of 7 mm. Hg or more evidence of pathologic condition
Pilocarpine	1928	Van Hofe	Use of miotics	Lowering tension to subnormal in glaucoma
Fluorescein	1922 1947	Theil Amsler and Huber	Fluorescein visible in pupil —with slit lamp	Indicative of increased capillary permeability
Vasodilator	1946	Gallois	Blood cholinesterase content	Nicotinic acid 0.03 or benzylimiazolin 0.025 g. orally. After an hr. no decrease in pressure
Vasculat (Lability)	1952 1953	Leydhecker Leydhecker	Subconjunctival injection of 0.3 ml. of sol. novocaine 1%, 1.0 ml. Vasculat (butyl Sympatol)	Increase of 12 mm. Hg pathological 40% cases showed + water-drinking test 89% showed + in Vasculat test
VASCULAR OR BULBAR COMPRESSION				
Massage	1878 1911 1912	Pagenstecher van Gelder Knapp	Fall of ocular tension after massage of globe	1 min. massage of globe or 250 gm. weight on closed lid for 10 min. Glaucoma—tension falls less and returns to normal 30 min.
Decubitus	1918 1922	Kollner Thibert	Head low for one hr.	Rise of 6 mm. Hg indicates glaucoma
Venous congestion	1925 1929 1930	Theil Schoenberg Wegner	Constricted neck with bandage or by sphygmomanometer cuff	Rise of 6 mm. Hg after 1 hr. indicates glaucoma
Tonography	1934 1950	Bock Gradle and Stough Moses and Bruno	Corneal massage with Schiøtz tonometer	15 gm. for 2 min. Glaucomatous eyes show less drop in tension
Bulbar	1950 1950 1951	Bietti Grant Suda and Kamao	Compression by dynamometer	Pressure for 10 min. enlargement of blindspot

TABLE 1—(*continued*)

Test	Year of Report	Various Workers	Based On	Interpretation
Bulbar	1953	Blaxter		Tonometer 15 gm. plus 50 gm. applied with dynamometer
Lability or pressor congestion	1945	Bloomfield and Lambert	Combination or cold-pressor test of Hines and Brown with jugular compression of Wessely and Schoenberg	Rise in ocular tension over 9 mm. Hg. Positive in rising phase diurnal variation; falling negative
Vacuum cup	1946	Herzau	Suction	Normal eyes drop in tension after test
Goniodynamometric	1949	von Beuningen	Pressure with dynamometer	Pressure of 100 gm. for 4 min. after which blood enters Schlemm's canal—disappears sooner in glaucoma than normal
Gonioscopic and Roenne's colloidometer	1951	von Beuningen		Slightly higher density in glaucoma
Dynamometer	1951	Kleinert and Grun	Maximum compensation	Blood escapes into aqueous veins Glaucoma—100 gm. required Normal—more than 150 gm. required
			SURGERY	
Paracentesis or anterior-chamber puncture	1930–1933	Kronfeld	Anterior-chamber puncture	Normal—25–35 mm. Hg. Glaucoma 40–60 mm. Hg.
			SENSORY	
Dark-room	1910 1922 1949 1953	Gronholm Seidel Kronfeld Higgitt	Dark-room for one hr.	Variation over 6 mm. Hg. pathognomonic
Dark adaptation	1926 1952	Derby, *et al.* Van Wien	light threshold and dark adaptation	Dark adaptation defective in glaucoma
Reading	1931 1949	Gradle Kronfeld	Read small print concentratedly for 45 min.	Rise of 10–15 mm. Hg
Flicker fusion	1949	Miles	Length of latent period and ability of cones and their pathways	Defective flicker fusion frequencies in glaucoma

ocular hydrostatic pressure due to vascular "erection" of the uveal tissue from neurovascular causes, increase in the episcleral venous pressure or in the vortex veins, impediment of flow through the pupils, the suction effect of the canal of Schlemm, failure of the constrictive ability of the ciliary arterioles, a disturbance of the central mechanism (wherever that may be) for the control of the intraocular pressure, increase in the size of the lens with age, pupillary dilatation in a dark-room or the result of emotional disturbances, and minor ocular trauma.

Most convincing is the fact that acute rises of intraocular pressure follow drug mydriasis in eyes with narrow angles; whereas, the pupils of glaucomatous eyes with open angles and normal width can be dilated for the most part with impunity.

Again, and most convincing, is that a peripheral iridectomy, performed before the development of peripheral anterior synechias have occurred to a large extent, will prevent in a large percentage (over 90) of cases a recurrence of the acute attack. The glaucoma is cured. Weinstein, for example (1954), states that of 865 cases of acute glaucoma seen in 20 years only four developed an attack of acute glaucoma in the iridectomized eye.

The exact mechanism of how an iridectomy works as a cure for acute glaucoma has been the subject of considerable debate, often heated, since the day when von Graefe announced his discovery. Even today the followers of the "neurovascular" school argue that the iridectomy is curative through its effect on the neurovascular reflexes of the eye.

As Scheie has pointed out, this is hard to accept when the great extent of the vascular bed of the eye is considered. Furthermore, dilatation of the pupil and engorgement of the vessels in the posterior uvea do not cause a rise in pressure when pupillary and angle block have been eliminated in the periphery of the iris. Also in severe uveitis, when the ciliary body is obviously engorged, there is usually hypotony, unless the angle is blocked with debris.

In addition, in cases of acute congestive glaucoma, the classical symptom of halos is the result of epithelial edema of the cornea produced by a sudden increase (qualitative) of intraocular pressure with swelling and congestion of the ciliary body.

The cases mentioned by Duke-Elder where ocular tension may be found to be 70 mm. Hg or above, without congestion, pain, or particularly halos, I think we can consider as instances where the angle is blocked and the resistance to aqueous outflow is markedly and acutely increased, but that for some unknown (as yet) reason the histamine induced engorgement of the ciliary body or uveal tract has not occurred. Might this not therefore be considered as further evidence that blockage of the angle precedes the "erection" of the uvea?

On the other hand, it is fair to state that many notable observers including Duke-Elder "agree with Troncoso that, in congestive glaucoma, the tension of the eye may be controlled postoperatively, even although the iridectomy is obviously not basal, the angle remains gonioscopically closed, and artificial drainage cannot be elicited clinically."

It is necessary, I think, to find the answers to the following questions:

1. Why do our patients, whose intraocular pressure has been normalized as the result of iridectomy, apparently show no further evidence of a neurovascular disorder within the eye that can be clinically observed or measured?

2. Do those of us who have normal anterior chambers ever show any condition that would have produced an acute glaucoma had our angles been narrow?

3. Do people, as the result of psychogenic or emotional factors, have edema or congestion of the ciliary body insufficient to precipitate an attack of acute glaucoma in the presence of normal angles?

4. Has one of us ever seen an attack of acute glaucoma precipitated in one's office by the use of a mydriatic in patients who do not have uveitis or a narrow angle? Posner and Schlossman, discussing the syndrome of glaucomatocyclitic crises, say "any case of acute glaucoma in which the eye does not have a narrow angle, or in which the angle appears open, should be watched by daily slitlamp studies for the appearance of keratic precipitates."

These are only some of the questions that need further study and suitable answers. Controversy here is good and let us have more of it.

As the disease of acute congestive glaucoma progresses the attacks become more frequent, begin earlier in the day, and last

longer. The variation between the highest and lowest pressure tends to lessen as the base pressure rises. As time goes on the disease becomes comparable to simple glaucoma in changes in the field of vision, optic atrophy, and cupping. No or very little episodic variation of tension is encountered and there often is no congestion, for compensation occurs, just how is still speculative.

On the other hand, late cases of simple glaucoma may develop halos with blurring of vision, and if the angle of the anterior chamber is narrow, as it often is in the aged, peripheral anterior synechias are produced.

It is the convergence of the two forms into the final common denominator that has led to confusion, for in the absence of a most careful history it may be quite impossible to say whether or not this patient has congestive or simple glaucoma (Miller).

Diagnosis

It is difficult to determine the incidence of primary glaucoma in the general population. This is because many studies have included both forms of primary glaucoma together, but chiefly because it is so difficult especially for the general practitioner and the optometrist (sight-testing optician) to recognize early cases of chronic simple glaucoma. However, from the study of statistics gathered by careful observers it is believed that at least two percent of the general population have primary glaucoma, and that approximately 25 percent of the cases of blindness are caused by primary glaucoma.

There is a great need to improve our methods of the early diagnosis of glaucoma, especially for the little fellow working alone in his office. It is encouraging to note that our thoughts are constantly being directed to this goal and, as a result, our methods have improved, especially in the last 10 years. These are:

1. Greater awareness of the disease occurring in patients over the age of 40 years and the need for a thorough examination by an ophthalmologist. Braley, for example, found that one out of every 40 persons past the age of 40 years had undiagnosed glaucoma.

2. The establishment of special clinics in various places throughout the world, and the growing interest in these special clinics or centers as a measure to control and supervise properly the treatment of patients with primary glaucoma. These clinics have shown their great value in permitting careful study of this baffling problem and in the teaching of residents, interns, and special visitors. There should be more of these centers everywhere.

3. The increasing use of routine tonometry in all patients, aged 40 years or over, using tonometers that have been checked for accuracy and standardization. The electronic tonometer has proved to be of great value. It has a high degree of accuracy. Friedenwald and others emphasize the importance of recording the tonometric readings with two different weights in order to rule out discrepancies in ocular rigidity which might interfere with accuracy of determinations. This is particularly important in myopia.

4. The careful study of the anterior chamber with slitlamp and gonioscope. A narrow angle is particularly suspect and all patients over the age of 40 years with a narrow angle should be given the darkroom test. Kronfeld found that patients with narrow angle get glaucoma in 80 percent of the cases within five years.

5. Tonography, using either the method improved by Grant or the bulbar-pressure test (Blaxter) which is easier to perform by the ophthalmologist working along. Tonography is of particular importance in chronic simple glaucoma.

6. Perimetry, in chronic simple glaucoma, remains one of our most important measures of early diagnosis and follow-up.

7. Ophthalmoscopy, in chronic simple glaucoma, particularly. This needs no com-

ment except to emphasize that a careful record or drawing of the disc should be made in all persons past 40. The classification devised years ago by Elschnig is a valuable method of notation.

8. The study of the diurnal phasic variation in ocular tension in chronic simple glaucoma. This cannot be easily done as a routine, but it is possible to arrange to take the ocular tension at different times of the day in one's office, in suspected cases, without too much inconvenience to the patient.

9. Provocative tests are given in Table 1. Table 1 is not complete in all respects but is given to indicate the various directions of thought along this line. There are many investigators who are interested in this phase of the subject. Their reports show many areas of disagreement and interpretation. No doubt new provocative tests or combinations of old ones will be developed in the future.

At present the most reliable provocative tests seem to be:

a. The waterdrinking test (Schmidt) in chronic simple glaucoma, since this test is independent of the rising or falling phase of ocular tension. It may well be that the subconjunctival injection of Vasculat (butyl Sympatol), a strong vasodilator, recently described by Leydhecker, may be of greater value. Leydhecker found that 89 percent of the glaucomatous eyes responded, after this injection, by a rise in pressure of more than 12 mm. Hg.

b. The dark-room and mydriatic test in congestive glaucoma.

10. Above all, however, and as a fundamental basis, is the taking of a most careful and painstaking history. Kronfeld's paper on "Diagnosis," is particularly noteworthy of emphasis in this discussion, because of its common sense.

11. Finally, I should like to emphasize that the patient should not be informed that he has glucoma until the diagnosis is definitely certain. I have seen a number of patients who had been told that they had glaucoma, sometimes after a single tonometric reading. I was only convinced after most careful and repeated studies over a long period of time that they did not have glaucoma. Such patients are hard to reconcile and continue to live in a state of mild neurosis between visits for reassurance. It seems, sometimes, that a patient is worse off if he thinks he has glaucoma when he hasn't, than when he knows for certain that he has it.

Summary

1. Seventy-five years ago the essential clinical facts regarding primary glaucoma were well known and described, but the problem of its etiology has, to this date, been unresolved. However, in the past 10 years, especially, much scientific information has been accumulated, particularly along the physico-chemical lines prophesied by Duke-Elder 25 years ago.

2. We know much about how the aqueous is formed and eliminated, and a great deal about the physico-chemical mechanism of the maintenance of normal intraocular pressure.

3. We suspect that the control of the normal intraocular pressure is a function of the brain, presumably the hypothalamus. As one evidence for this belief there is the interesting and curious behavior of the patterns in the normal individual of the bilaterally synchronous diurnal variations of intraocular pressure and its relationship to the blood supply to the uvea, particularly in the ciliary body.

4. Primary glaucoma is a complexity of diseases whose chief symptom is the elevation of the intraocular pressure. It is manifested in two forms which are probably entirely separate disease entities.

5. Chronic simple (open-angle) glaucoma is characterized by rhythmic wide and uncontrolled swings of the diurnal intraocular pressure, without other obvious clinical signs until the disease is far advanced. It may occur without abnormal elevation of pressure (low-tension glaucoma).

Disease of the blood vessels resulting in disturbance of the arterial but especially the

venous pressure is undoubtably a factor in its etiology and especially so in its evolution. The maintenance of adequate nutrition, notably to the optic nerve, ciliary body, and retina, is increasingly affected as the disease progresses, often in spite of a successful filtration operation.

In addition, there is increasing and impressive scientific evidence that resistance to the outflow of aqueous is always present and increases with the advancement of the disease. For this reason there exists the laudable controversy between the neurovascular and the mechanical schools of thought. Whether there exists also a disease or lesion in the "control center" in the brain, wherever that may be, is as yet unproved.

6. Acute inflammatory (narrow-angle) glaucoma is, I firmly believe, an entirely different condition, for the reasons already outlined. Here there is undoubtedly a strong neurovascular and psychogenetic background, but the narrow angle and its ease of closure is primarily the site involved.

There is good evidence that the narrow angle of affected individuals in an hereditary and familial, anatomic and architectural feature. As such, any mechanical factors existing within such an eye, as well as a neurovascular crises, are capable of precipitating an acute attack. I suspect that in many cases the neurovascular crisis of uveal erection is the result and not the cause of the acute closure of the angle.

Whether or not this is true for all cases is for future studies to decide. However, I hope that the evidence that I have presented is strong enough to convince you all that acute inflammatory glaucoma is a disease entity, entirely separate in all of its factors, except elevation of intraocular pressure, from chronic simple glaucoma.

7. If such a concept can be agreed upon, the way for future studies is more clear, for in my opinion the confusion in the past between these two entities has been a great hindrance to the understanding of these two ocular diseases and the proper and suitable medical and surgical treatment of each.

8. The advancement of our knowledge, as the result of modern scientific facilities and apparatus, has helped us enormously in our approach to the fundamental problem of early diagnosis, particularly by the ophthalmologist who has to work alone in his office practice. More thought must be given to this important phase of the problem.

9. The prospects for solving the thorny problems of the primary glaucomas, in the light of modern trends of thought and work, are exceedingly bright and hopeful.

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A NEW TYPE OF APC VIRUS FROM FOLLICULAR CONJUNCTIVITIS*

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The adenoidal-pharyngeal-conjunctival (APC) group of viruses was discovered by Rowe et al.[1] who isolated them in cultures of adenoids and tonsils. They comprise a large group of viral agents which can be subdivided into serologic types by means of specific neutralization tests.[2] Type 4 was originally isolated and described as RI 67 virus by Hilleman and Werner[3] and is now established as the cause of acute respiratory disease (ARD) in military recruits.[4] Bell et al.[5] have established the role of Type 3 APC viruses as etiologic agents in epidemic outbreaks of "pharyngoconjunctival fever." Ryan et al.[6] have described the clinical characteristics of eye infections due to APC virus Types 3 and 4. Types 3, 4, and 7 have been recovered from military recruits suffering from respiratory illnesses ranging in sever-

* From the Departments of Microbiology and Ophthalmology and the Francis I. Proctor Foundation for Research in Ophthalmology, University of California Medical Center. This study was supported in part by a grant (B-604) from the National Institutes of Health, Bethesda, Maryland.

ity from mild febrile rhinitis to primary atypical pneumonia.[7] Types 1, 2, 3, and 5 have been isolated from sporadic cases of nonbacterial pharyngitis[4,8] and are frequently found in adenoid and tonsillar tissue removed at surgery.[2] Type 8 has been recovered from a case of epidemic keratoconjunctivitis and may be etiologically related to the disease.[9]

To date APC virus Type 6 has not been encountered in clinical illness, and it has been isolated only once from adenoid tissue in tissue culture.[2] It is the purpose of this communication to report two cases of clinical eye infections from which Type 6 was isolated and in which the patients demonstrated by their specific antibody response that it was the etiologic agent of their illness.

Case reports

Case 1

Dr. H. M., a 26-year-old physician, was seen on May 2, 1955, complaining of conjunctivitis with a "foreign-body sensation" in the left eye for 24 hours. There was a "pea-sized" left preauricular lymph node noted by the patient on May 2nd, but no discharge from the eye. There were no symptoms of systemic illness and no respiratory symptoms.

Examination showed a tender, enlarged left preauricular node, 20/20 vision, normal corneal sensitivity, and normal lids. There was marked tearing, and congestion of tarsal and bulbar conjunctiva with intense follicular hypertrophy in the lower fornix. The cornea was clear, and there were no precipitates, flare, or cells in the anterior chamber. General physical examination was within normal limits.

Bacteriologic culture of the lids showed occasional nonhemolytic Staphylococcus albus, and cultures of the conjunctiva were sterile. Smears from the conjunctiva showed some mononuclear cells, no polymorphs or eosinophils, and no bacteria.

Scrapings taken from the conjunctiva were inoculated into twice-washed HeLa cell cultures (obtained from Tuskegee Institute), and a maintenance medium of 10-percent chick serum in mixture 199 was added. After five days of stationary incubation at 36°C., cytopathogenic effects were noted; cell degeneration was almost complete by the seventh day. The virus was passed in HeLa cell cultures. Chorioallantoic membranes inoculated with the same scrapings showed no lesions.

Clinical course. The inflammation of the eye did not change materially for several days, and then the conjunctivitis subsided gradually, leaving no residuum whatever except for conjunctival edema persisting for two weeks. The cornea remained clear. The preauricular node remained enlarged for three weeks but was nontender after the first few days of illness.

On May 8th, the patient developed symptoms of systemic illness, with fever of 101°F., muscle pains and aches, and moderate prostration, but no respiratory discomfort. The febrile illness lasted two days and then subsided completely. Since May 10th the patient has been entirely well.

Past history. Good general health. Herpes zoster on buttocks two years ago. No history of herpes simplex. As an interne at the San Francisco County Hospital, the patient examined many individuals with "red eye," usually considered due to a foreign body. His wife and child developed no manifest infection.

Virus studies. The earliest cytopathogenic effects in HeLa cells were noted on the fifth day after inoculation of the scraping. Degeneration was virtually complete on the seventh day of stationary incubation. The type of degeneration was compatible with the type seen with APC viruses. After five passages in HeLa cells, the virus reached a titer of $TC50 = 10^{-5.4}$, and cytopathogenic effects were complete five days after inoculation of 100/TC50.

Through the courtesy of Lieut. Col. T. O. Berge, 6th Army Area Medical Laboratory, the virus was typed by neutralization test and shown to belong to Type 6 of the APC

group. This typing was confirmed in the laboratory of Dr. R. J. Huebner. The virus fixed complement with antiserum to APC viruses, Types 4 and 8.

Neutralizing antibody titers. Neutralization tests in HeLa cells were performed with 1,000 TC50 in the usual manner, with a one-half hour incubation of the virus-serum mixture at 22°C. The patient's serum of May 2, 1955, did not neutralize the virus in 1:10 dilution. The patient's serum of August 9, 1955, neutralized the virus in 1:80 dilution.

Case 2*

Dr. C. M., on April 27, 1955, developed sensation of scratchiness in both eyes with mild scleral injection. Accompanying this, he had an "annoying sensation of fullness in the tissues surrounding the pharynx." The sensation was not that of a sore throat but was described as being vague and difficult of description. Approximately one week prior to onset of C. M.'s illness, his wife had developed an identical illness, and during the week between the two cases their four children, ranging in age from four months to five years, had had very similar illnesses. All of the cases had bilateral injection of the bulbar conjunctiva, with dried mucus on the lid margins in the morning. The oldest child complained of his neck and throat. None of the cases felt febrile; however, temperatures were not taken in any of the cases. The duration of the disease in the children was three to four days, and in the adults two to three days. In no case was the illness incapacitating, and the doctor continued to work throughout the period of illness. There was no known exposure of any of the cases to any similar cases outside the home, and no secondary cases were observed in the neighborhood.

Type 6 APC virus was isolated at the National Institutes of Health from the right eye of Dr. C. M. on the second day of illness. Neutralizing antibody titers to Type 6 were: The patient's serum of November 24, 1954, did not neutralize the virus in 1:4 dilution. The patient's serum of June 23, 1955, neutralized the virus in more than 1:16 dilution.

Eye and throat swabs obtained from the children during early convalescence were negative in HeLa cells.

* Information kindly furnished by Dr. W. P. Rowe and Dr. R. J. Huebner.

Discussion

The incidence of neutralizing antibodies against APC Type 6 in the general population in the Washington, D.C., area was found by Huebner et al.[2] to be about 30 percent. The virus isolated from Case 1 was employed in a serologic survey of persons, aged nine to 44 years, living in California. Twelve of 36 sera (33 percent) obtained between 1952 and 1955 had neutralizing antibodies to Type 6 APC virus in a dilution of 1:10 or greater. The majority of these sera were from persons over the age of 25 years, and most positive sera were also in that age group. APC virus Type 6 was also neutralized by one lot of commercial gamma globulin in a dilution of 1:80.

Thus, there is serologic evidence in at least two separate regions of the United States (Eastern Seaboard, California) that one third of the population may acquire infection with APC virus Type 6 before the age of 45 years. It is difficult to know whether the infection is asymptomatic in most instances, or whether a relatively mild and transient disease occurs frequently as illustrated in the two cases reported here. Since infection with an APC virus occasionally results in heterotypic antibody response, it is possible that some of the neutralizing antibodies represent a response to infection with other, more prevalent types.

In spite of the frequent association of APC virus infection with respiratory disease it appears possible that the conjunctiva frequently serves as a portal of entry for APC viruses. Conjunctivitis may occur alone, or may precede the onset of pharyngitis or systemic symptoms. It has also been observed

that experimental inoculation is more likely to result in objective illness if the virus is introduced by swabbing the conjunctiva, rather than by dropping or spraying it into the respiratory tract.[8]

Summary

APC virus Type 6 has been isolated from two patients with follicular conjunctivitis and associated mild systemic or respiratory symptoms. This is the first time that this virus type has been shown to produce human illness. Data are also presented on the distribution of this agent in a sample population and on the possible role of the eye as portal of entry for this infection.

University of California Medical Center (22).

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CHOLESTEROL-CONTAINING GRANULOMA OF THE ORBITAL WALL*

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Recently, a patient was referred to me with proptosis of the right eye caused by a large destructive lesion in the fronto-orbital bones. After investigation, this patient was operated on by Dr. Wilder G. Penfield at the Montreal Neurological Institute, with some unusual pathologic findings. The lesion was of a granulomatous nature, containing many cholesterol crystals mixed with old blood. A few weeks later a second patient was admitted to the Montreal Neurological Institute with the same type of lesion. A review of the records at the institute disclosed the occurrence of still another some years previously.

The clinical, radiologic, and pathologic characteristics of these three patients were essentially identical. A search of the literature has uncovered reports of only 12 other cases. It is proposed to present our three cases, to review the 12 others, to summarize their clinical and pathologic features, and to discuss their pathogenesis.

* From the Departments of Ophthalmology of McGill University and of the Royal Victoria Hospital. Read before the annual meeting of the Canadian Ophthalmological Society held at Bigwin Inn, Ontario, June 15, 1955.

Case reports

Case 1

The first patient, and the one who first

Fig. 1 (Nicholls). *Case 1.* Showing swelling in temporal region of right supraorbital ridge, with proptosis and displacement of the right eye nasally and downward.

consulted me, was a 46-year-old man, who presented himself in October, 1953, complaining of right proptosis and diplopia of about six weeks' standing. There was no pain or blurred vision. He stated that at about nine years of age he had received a blow on the head resulting in a left frontal scalp laceration. For many years before consulting me, his friends had noticed a fullness of the right fronto-orbital region. This was corroborated by photographs. A few weeks before this consultation he accidentally struck this region on a door, following which proptosis of the right eye and diplopia developed.

Examination disclosed definite fullness of the right supraorbital ridge (fig. 1). Exophthalmometer readings showed that the right eye protruded about three mm., and it was displaced slightly downward and inward. A firm mass fixed to the bony wall could be felt in the region of the lacrimal gland, but it was placed too medially to be perfectly typical of a lesion of this structure. The right superior rectus muscle was definitely limited in action. The patient had full vision in each eye and the fundi were normal. The pupils were regular, equal, and active to light and accommodation.

General physical examination disclosed nothing abnormal. Blood serology and chemistry were negative. The serum cholesterol level was 22.0 mg. percent. X-ray films showed an excavation of the base of the zygomatic process of the right frontal bone which extended slightly into the zygomatic bone (fig. 2). This cavity appeared to be in the diploe. The outer table was greatly thinned and displaced outward. The lateral portion of the orbital roof was rarefied and displaced downward. In only a few areas was compact bone observed between the lesion and the surrounding normal diploic spaces. The medial end of the cavity came close to the frontal sinus but was definitely separated from it.

Operation. On October 22, 1953, Dr. Penfield exposed the right frontal region by reflecting a right frontal scalp flap. Over the lateral aspect of the right supraorbital ridge the bone was eggshell thin. This was opened and dark brown fluid escaped revealing a cavity filled with soft yellow-gray, necrotic, amorphous material. The inner table of the frontal bone had been eroded exposing a very small area of dura, which was intact but yellow-stained. There was an erosion of the roof of the orbit through which the contents of the cavity extended, pushing aside and staining but not invading the orbital fascia. This whole area of bone was removed including a portion of the supraorbital ridge and the roof of the orbit; no plastic repair was attempted. Cultures of the soft amorphous material were sterile.

The postoperative course was uncomplicated. The proptosis of the right eye receded rapidly and the diplopia decreased. At the last examination, a year later, there was no proptosis. Full ocular movements had returned and there was no diplopia.

Case 2

The second case was that of a 40-year-old man who entered the Montreal Neurological Institute in January, 1954, complaining of a slight prominence of the left eye and diplopia for about two weeks. He had no pain or headache. He could not remember ever having received any injury to the left orbital region.

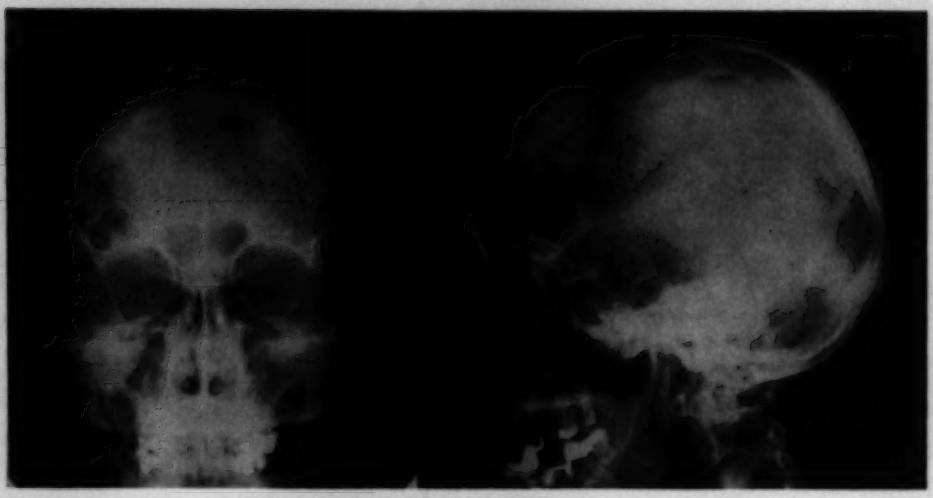

Fig. 2 (Nicholls). *Case 1.* Posteroanterior, and right lateral X-ray pictures of skull showing the osteolytic lesion in the right supraorbital ridge, frontal squama, and orbital roof.

On examination the patient was found to have very slight proptosis of the left eye which also was displaced slightly downward and medially. Diplopia occurred only in the extreme left upward gaze. No unusual mass could be felt in the orbit. There were no other visual disturbances. The pupils, visual acuity, fields, and fundi were normal.

General physical examination disclosed nothing abnormal. Blood chemistry and serology were normal. The serum cholesterol was 245 mg. percent. X-ray films of the skull showed an area of destruction similar to that described in the first case, but here it involved the later half of the left supraorbital region and adjacent portions of the frontal squama of the orbital roof (fig. 3). This area was surrounded by a thin intact layer of compact bone. It was not related to the frontal sinuses.

Operation. On January 29, 1954, Dr. Wilder Penfield operated on the patient, exposing the lesion through an incision in the lateral part of the eyebrow. Again paper-thin bone was found, the most marked areas being in the frontal squama just above the supraorbital ridge, and in the frontal bone just behind the zygomatic process. The cavity was entered through the attenuated area of the latter portion, and a small amount of dark brown fluid escaped. The cavity contained soft, yellow-gray amorphous, gritty material and a small amount of watery fluid containing crystals. No capsule separated this from the bony walls. Most of the roof of the orbit had been destroyed by the pressure of the mass. The latter was in contact with the orbital fascia, which was compressed and stained but not invaded, and with a portion of the dura in the frontal region, which was stained yellow but intact. The material was curetted away completely and the bony defect was closed, using a piece of tantalum. The patient made an uneventful recovery with complete subsidence of the proptosis and diplopia.

Case 3

Examination of the records of the Montreal Neurological Institute disclosed that a 57-year-old man had been admitted in July, 1944, with a lesion similar to those already described. On admission he complained of a right supraorbital swelling, mild vertigo, and diplopia.

He stated that, in 1919, he had been struck

on the right supraorbital ridge by a metal spike. A little later he developed a small painless swelling. Following an automobile accident in 1943, he entered the Montreal General Hospital for treatment. There was evidence of recent injury in the right orbital region, and at certain points over the supraorbital ridge the bone could be compressed and crepitus felt. The right eye was slightly proptosed and its upward movement limited. X-ray films showed a comminuted fracture of the lateral and superior margins of the right orbit with lateral displacement of the fragments. A biopsy was performed and the patient discharged. He did not return to the hospital. However, six months later the mass began to enlarge rapidly and the patient was referred to the Montreal Neurological Institute. On this admission the examination showed a firm irregular reddish swelling over the lateral margin of the right supraorbital ridge. The right eye was proptosed and displaced downward and medially. Diplopia occurred on looking upward. The visual acuity, fields, and fundi were normal.

General physical examination disclosed nothing abnormal. Blood serology and chemistry were normal. Serum cholesterol was not determined. X rays showed a large rarefied region in the roof of the right orbit involving the lateral three-quarters of the supraorbital ridge (fig. 4). The margins of the defect were relatively distinct and the lesion did not communicate with the paranasal sinuses.

Operation. On July 28, 1944, Dr. G. Morton operated, approaching the area through a right frontal scalp flap. The lesion was exposed after removing what remained of the supraorbital ridge. A large cystic mass involving the right frontal bone and the zygomatic process was found. It extended quite a way into the orbit and was adherent to the lacrimal gland, the orbital fascia below, and the dura above. None of these structures was invaded. It was soft, brownish, avascular, and contained cysts filled with yellowish glistening gelatinous material. There was a large supraorbital bone defect which was not repaired. The patient made an uneventful recovery with no recurrence. The diplopia and proptosis rapidly subsided.

Microscopic pathology

The histologic findings in all three cases were so similar that they may be described together. The soft yellowish material was composed of a fibrous granulation tissue

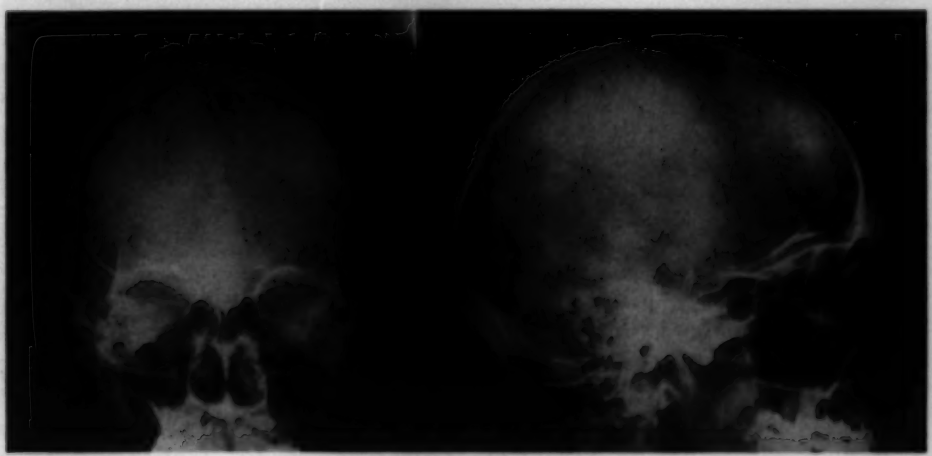

Fig. 3 (Nicholls). *Case 2.* Posteroanterior and left lateral X-ray pictures of skull showing the osteolytic lesion in the left supraorbital ridge, frontal squama, and orbital roof.

Fig. 4 (Nicholls). *Case 3.* Posteroanterior and right lateral X-ray pictures of skull showing the osteolytic lesion in the right supraorbital ridge, frontal squama, and orbital roof.

matrix (fig. 5) containing many sharp pointed clefts of a type usually associated with cholesterol crystals (fig. 6). Many multinucleated foreign body giant cells were seen along the margins of many of these clefts (figs. 7 and 8). The clefts occurred also in areas of complete necrosis.

There were many areas having no clefts. These were composed of a much more vascular stroma containing many mononuclear cells. Some of these latter cells contained a large amount of eosinophilic cytoplasm in which were many cellular debris and particles of blood pigment (fig. 9). Frequently, the same type of cell was seen to have a large, clear vacuolated space in its cytoplasm (foam cells) (fig. 10). Occasionally two nuclei were seen in this latter variant. Both the eosinophilic and foam cells were seen to be concentrated about blood vessels. No necrosis occurred in these highly vascular areas, but it was abundant in regions remote from the blood vessels.

Small fragments of bone were scattered

Fig. 5 (Nicholls). *Case 2.* Phosphotungstic and hematoxylin, ×170. Fibrous tissue and reticulum.

Fig. 6 (Nicholls). *Case 1.* Phosphotungstic and hematoxylin, ×170. Fibrous tissue and reticulum, crystal clefts.

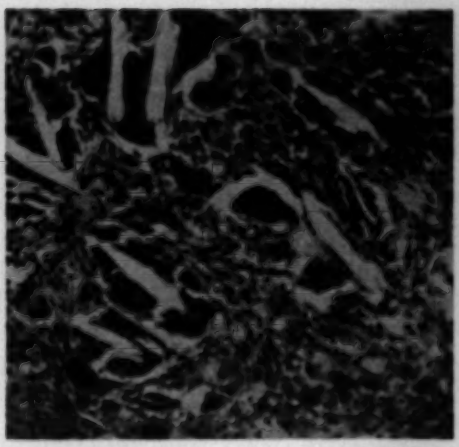

Fig. 7 (Nicholls). *Case 2.* Hematoxylin and eosin, ×450. Crystal clefts and giant cells.

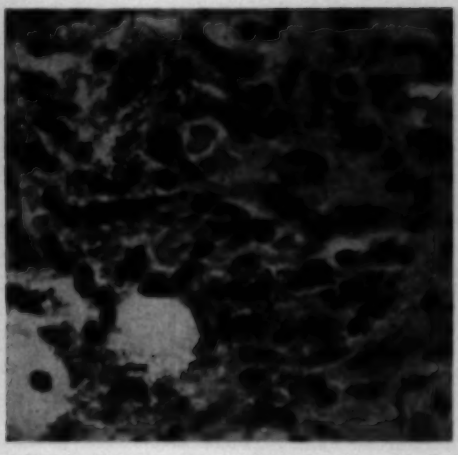

Fig. 9 (Nicholis). *Case 1.* Hematoxylin and eosin, ×1,300. Phagocytic cells, many containing hemosidirin granules and foam (or xanthoma) cells.

throughout the tissue and frequently were associated with the multinucleated giant cells.

Yellowish-brown hemosiderin granules and crystals of other blood pigments were seen in all regions, both extracellularly and within the cytoplasm of the eosinophilic cells. Scherlach-R staining demonstrated a large amount of neutral fat, some within cells, but usually lying free in the tissue spaces. The cavities of the foam cells could not be stained.

There were several small areas of dense inflammatory infiltration. These contained mostly lymphocytes and polymorphonuclear leukocytes. In the first two patients a small number of eosinophiles could be seen widely scattered throughout the tissue. These foci of leukocytes frequently were associated with the areas of necrosis.

Bands of fibrous connective tissue extended through the tissue, varying in amount in different areas, but being most abundant

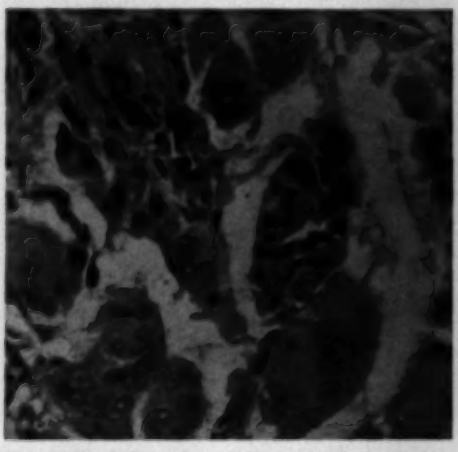

Fig. 8 (Nicholls), *Case 2.* Hematoxylin and eosin, ×1,300. Giant cells and crystal clefts.

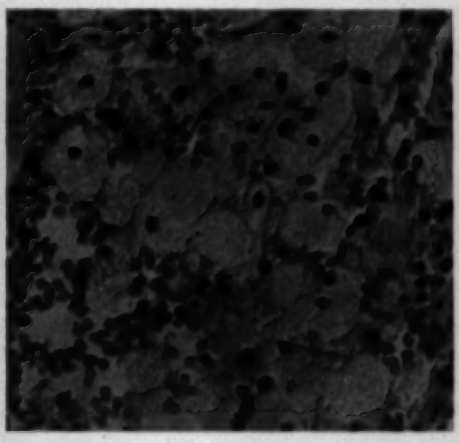

Fig. 10 (Nicholls). *Case 1.* Hematoxylin and eosin, ×600. Foam (xanthoma) cells and reticulocytes.

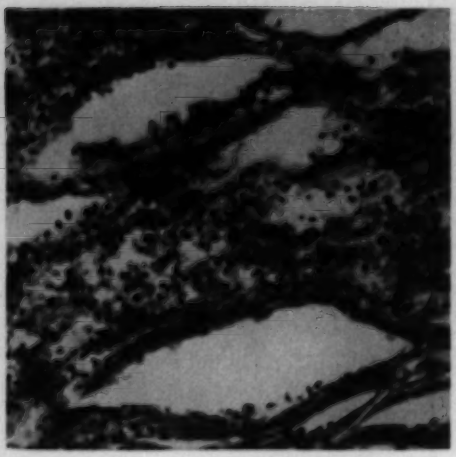

Fig. 11 (Nicholls). *Case 3.* Laidlaw's connective tissue stain, ×600. Collagenous connective tissue fibers about crystal clefts with small round-cell infiltration.

in the periphery (fig. 11). Fibrosis also was abundant in the region of the clefts. It was most delicate in the area of greater vascularity. Here and there a fine reticulum could be seen, particularly in the region of the foam cells (fig. 12).

Sections of the bone taken from the margin of the lesions showed infiltration of the marrow spaces by connective tissue containing both spindle and stellate cells. Greater numbers of round or oval cells were arranged in rows along the margin of the bony trabeculae. They probably were osteoblasts. No mononuclear, eosinophilic, or foam cells, or crystal clefts were found along the bony margins.

Discussion

Hanbery and Rayport[1] in a detailed study of these cases have made an exhaustive investigation of the medical literature in an attempt to discover others, but they were able to discover only six authentic cases of similar type. I am greatly indebted to these authors for their generous help in preparing my report.

Pincus,[2] in 1933, described two cases very similar to ours and referred to them as pseudocholesteatomas. The clinical, X-ray, and pathologic findings in his two cases were identical. Unfortunately, he reported microscopic studies in only one patient. However, this one presented a histologic picture identical with that in our three patients, and with certainty can be included with our series.

Knapp,[3] in 1934, reported two cases of exophthalmos due to an expanding frontoorbital lesion, which he thought were examples of xanthomatosis. The clinical, X-ray, and pathologic characteristics of the local lesion in both cases were similar to ours. But his first patient had an affection of the optic nerve and his second had a positive Wassermann and a serum cholesterol value of 300 mg. percent.

Kreibig,[4] in 1938, described a case of unilateral proptosis due to a "so-called cholesteatoma" of the supraorbital ridge. The characteristics of this lesion was very similar to ours.

In 1946, Hanbery[5] reported another case, which he included in a larger description of 12 cases of orbital tumors. This was in a young man, aged 31 years, who gave a history of pain over the right eye, proptosis,

Fig. 12 (Nicholls). *Case 2.* Laidlaw's connective tissue stain, ×1,300. Reticulum and histiocytes.

and diplopia existing for two months. A preliminary diagnosis of a bony growth behind the right eye was made and a course of X-ray therapy was given. There was some regression for awhile. Visual fields, fundi, and visual acuity were normal. Serum cholesterol was 178 to 195 mg. percent. Finally, local removal was carried out. The tissue removed was grossly and microscopically similar to ours. The patient made an uneventful recovery.

Ramsey and his confreres,[6] in 1948, reported two cases of unilateral proptosis and diplopia due to what they thought to be a posttraumatic granuloma of the orbit. There is some intermixing of their cases and ours. The first of their two is the same patient described as our third. Both their patients had a history of trauma and presented the typical clinical, X-ray and pathologic picture.

These same authors reported the occurrence of a lesion with the same radiologic and histologic characteristics, but occurring in the 10th left rib. This was in a 57-year-old man and was preceded by an injury. General physical examination disclosed no other abnormalities, the diseased area of rib was removed and there was no recurrence.

In 1953, Ingalls,[7] in his book on *Tumours of the Orbit*, described six more cases in a chapter entitled "Cholesteatoma." Later in this chapter he stated that his cases differed from the true cholesteatoma, being more like xanthomatous tumors. Clinically and pathologically they were so similar to ours as to make it certain that they are identical. Two of his patients were females, and thus he presents the only two females ever reported. His youngest patient was 37 years and his oldest 63 years of age. In one patient apparently the proptosis first occurred in childhood, but operation was not carried out until an age of 55 years. In one other, a man aged 63 years, the condition had existed for 20 years at the time of operation. The tumors were situated as in our cases, two being on the right and four on the left side. All showed the same proptosis, diplopia, X-ray, and histologic features. In four patients the vision of the affected eye was blurred, owing apparently to pressure on the optic nerve. No visual field studies were presented. In two patients the occurrence of proptosis was preceded by a frontal sinus operation—in one case by about six years and in the other by about seven years. In all cases the mass did not recur after removal. The proptosis and diplopia quickly subsided.

From a detailed study of our three cases and those reported in the literature it is possible to make the following summary of known facts, and to form an opinion of the pathogenesis of these lesions.

Age and sex

Of the 15 patients two were female and the remainder male. They ranged in age at operation from 28 to 63 years.

History of trauma

Ten of the 15 patients gave no history of injury in the orbital region. In three there was a definite history of trauma, and in two of these there was a history of two injuries. In two patients surgery had been performed on the frontal sinus of the same side several years previously.

Presenting symptoms

Proptosis and diplopia were the most common symptoms. Usually there was no pain, and when present it was not severe. Failing vision was recorded in five patients —Knapp's first case[5] and four of Ingalls'.[7]

Duration of symptoms

Symptoms were present for periods of a few weeks to many years. In our first case, swelling was noted in the supraorbital region for several years, and in our third case for a recorded 25 years. In both these patients the second accident involving the same region seemed to accelerate the development of the mass and the symptoms. In one of Ingalls'[7] cases the history went back about 50 years to childhood.

Signs

All the patients showed unilateral exophthalmos which usually was not marked. The eye was displaced down and to the nasal side. Each patient showed a limitation of up and outward gaze of the affected eye, and diplopia. At least 12 of the patients had a visible or palpable abnormality of the lateral portion of one supraorbital ridge. Seven of the patients were affected on the right side and eight on the left.

Visual acuity was affected in five patients. In Knapp's[3] case, there was an enlarged blindspot and a central scotoma, owing, as thought, to an optic neuritis. In Ingalls'[7,8] group, four had defective visual acuity. One, a diabetic, had pallor of the optic nerve suggestive of optic atrophy. But after operation the pallor disappeared. In two others there was papilledema, and in a fourth the fundus was normal. Unfortunately, he[8] was able to obtain a record of the postoperative visual acuity in only one patient. In this, the visual acuity improved but did not return quite to normal. It is logical to conclude that the various pictures described merely represent different manifestations of pressure on the optic nerve. Ramsey and his confreres described venous engorgement of the retina of one patient.

Laboratory data

In no case were there anemia, marked leukocytosis, eosinophilia, or elevated sedimentation rate. In Knapp's second case only was there a positive Wassermann. In one case only was a raised serum cholesterol recorded, and there it was only slight. This was in Knapp's[3] second case. But serum cholesterol levels were determined in only four patients.

X-ray findings

X-ray studies of the skull were made in 12 of the 15 patients and all were similar. In each instance a rather well-defined destructive lesion was present in the lateral portion of one supraorbital ridge. The adjacent frontal squama and the orbital roofs were involved to varying degrees. In several cases the destruction extended into the zygomatic process of the frontal bone. Ramsey and his confreres felt that in their second case the lesion extended into the greater wing of the sphenoid bone, but re-examination of the X-ray films does not support this view. In all other cases the lesions were confined to the frontal bone and were separate and distinct from the frontal and other sinuses. The lesions usually were separated from the normal diploic space by a thin line of compact bone. No other cranial or skeletal osteolytic lesion was found.

Treatment

In each of our cases at operation the abnormal tissue was removed locally. In only one—my first patient—was a craniotomy performed for an extradural approach to the floor of the anterior fossa. All the other lesions were exposed directly, either from below or above the supraorbital border, or through the temporal fossa. In each case the cavity was completely curetted. The orbital fascia was removed only in one patient, but otherwise the operation was limited to removing the granulomatous tissue. Though in several cases the dura was in contact with the lesion, there was no extradural extension of the abnormal tissue. The roof of the orbit was destroyed to a varying extent in each case causing displacement of the orbital contents.

X-ray therapy was attempted in one case only—Hanbery's.[5] A definitely beneficial effect followed the administration of 6,000 r, but symptoms returned after several months. Postoperative X-ray therapy was not employed in any case.

Results of treatment

All patients recovered rapidly after surgical removal of the lesion with complete subsidence of the proptosis and diplopia. Unfortunately, visual acuity was tested postoperatively in only one of the affected cases.

It improved without completely recovering.[8] As far as is known, the lesions never have recurred. Periods of observation usually have not extended beyond one year. However, in our first patient there has been no evidence of recurrence in two years. In our third patient no recurrence was seen at examination three years postoperatively, and he is alive 11 years later. Unfortunately, it has been impossible to examine him again. The second patient of Ramsey and his confreres[6] recently has been examined. He is without symptoms and has no clinical or X-ray evidence of recurrence. Knapp[9] recorded no recurrence in a 17-year follow-up of one of his patients. As far as is known none of the patients have developed another similar lesion anywhere else in the body.

Differential diagnosis

The clinical characteristics of the proptosis and of the tumor mass in these patients are indistinguishable from those produced by many other lesions. The X-ray findings considerably reduce the possibilities. But still one must consider such disturbances as neoplasm, inflammation, fibrous dysplasia, osteitis fibrosa cystica, postraumatic cyst, or any one of the several forms of histiocytic granulomas. Thus, in most patients the final diagnosis will not become clear until operation, or until microscopic examination has been completed.

If one were to consider the clinical, radiologic, and pathologic features of these cases, then the lesion must be differentiated from; (a) epidermoid and dermoid cysts, (b) giant-cell tumor, (c) diploic hematoma, and (d) from at least two of the three types of lesion which recently have been referred to collectively by Goodhill[9] as histiocytic granulomas, or as histiocytosis X by Lichenstein.[10] In this last, the two types of interest are eosinophilic granuloma, and Schüller-Christian disease.

It should be relatively easy to distinguish these lesions from dermoid and epidermoid cysts. In these latter tumors there always are remnants of the epidermis and associated structures. Many of these contain large amounts of cholesterol, so that Müller,[11] in 1838, referred to them as cholesteatomas. However, the presence of cholesterol is about the only similarity between them and our cases. Pincus[3] and Kreibig[4] referred to their lesions, which were similar to ours, as "pseudocholesteatomas," thus differentiating them from the true cholesteatoma.

Giant-cell tumors are exceedingly rare lesions in the skull, and are believed to occur only in those bones derived from the cartilage (Geschickter and Copeland[12]). Also, the histodogic characteristics of the giant-cell tumor are quite different from the present lesion. It is to be noted that Geschickter and Copeland[12] describe a giant-cell tumor in which there are giant cells, xanthoma or foam cells, and pigment containing histiocytes. But this variant of the giant-cell tumor is found only in extraskeletal areas and never in the skull.

Diploic hematoma

In five of the 15 cases discussed here, there was evidence of trauma antidating the lesion. In my first, the connection with trauma is uncertain as the fullness of the fronto-orbital region preceded a recent injury by many years. It is not clear from the history whether a previous injury in childhood actually brought about this fullness in the orbital region. In both of the cases reported by Dr. Ramsey and his confreres[6] there was a definite history of trauma. As already noted, their first patient is the same person reported in our present series as the third. In two of Ingalls' cases frontal sinus surgery had been carried out several years previously.

Ramsey and his confreres felt that trauma was the initiating cause in their patients. They felt that the essential lesion was a chronic granuloma, composed of foreign-body giant cells, fibrous connective tissue, phagocytes, and containing various blood pigments and cholesterol. They felt that

probably injury caused hemorrhage, which decomposed with the release of blood pigment and the formation of cholesterol crystals, which in turn produced a foreign-body granuloma. This certainly is an attractive theory. However, if this be the true course of events, then a history of trauma should be obtained more frequently. Also, injury about the face must be very common and if it be a cause, then this type of lesion should be much commoner than it apparently is. Further, the granulomas found in the five patients where there was a history of trauma, were identical with those found in the 10 patients where there was no trauma.

Histiocytic granuloma

Finally, these lesions should be thought of in relation to a rather poorly understood pathologic process recently called histiocytic granuloma by Goodhill.[9]

The knowledge of these lesions up to 1928 was reviewed by Rowland,[13] He collected 12 cases from the literature and added two of his own. He felt that the basic disturbance was in the lipoid metabolism which led to an excess of lipoids in the body fluids, resulting in a diffuse or nodular hyperplasia of the reticuloendothelial system. He felt that all the previously described xanthomatous lesions of soft tissues affecting such structures as the eyelids, tendon sheaths, and skin should be collected under the same heading, since they represent disturbed cholesterol metabolism. He also felt that Schüller-Christian's disease, which mainly affects bones, was another form of the disturbance. Thus, he brought it into relation with Niemann-Pick's, Gaucher's, and Tay-Sach's diseases. He suggested the general term of xanthomatosis to include all these disturbances of lipoid storage and metabolism which involve the reticuloendothelial system. He thought the lesions may be solitary or multiple, and may be precipitated by trauma, irritation, or infection.

As more cases were studied it became apparent that the majority of the patients with Schüller-Christian's disease did not have an elevated serum cholesterol, and thus the theory that it is based on an abnormal lipoid metabolism with hypercholesterolemia came into doubt. Most investigators now feel that this disease should not be included with the lipoidoses. They believe that only Niemann-Pick's disease, Gaucher's disease, and Tay-Sach's disease, and those xanthomatous lesions that are associated with hypercholesterolemia (xanthoma tuberosum multiplex) should be included in this group.

During the last few years many workers have come to believe that Schüller-Christian's disease is closely related to Letterer-Siwe's disease and eosinophylic granuloma. In all three bone lesions are a dominant feature. In this group of diseases there may be no hypercholesterolemia. Farber,[14] in 1941, originated the view that the three represent different phases of the same disease process, though this has not been fully accepted. Goodhill[9] agrees with Farber and has suggested the name of histiocytic granuloma for the group. Recently, Lichenstein[10] has reviewed the whole subject and has suggested the name of histiocytosis X. While injury and various infections have been suggested as possible etiologic factors, no good evidence has been presented to prove that this is necessarily so. It is safer for the present to consider them granulomatous lesions of unknown origin involving the histiocytes of the reticuloendothelial system, and to describe the three manifestations of the disease under the names of Letterer-Siwe's disease, eosinophilic granuloma, and Schüller-Christian's disease.

1. Letterer-Siwe's disease. This occurs in infants and children, and usually is rapidly fatal. It is characterized by lymphadenopathy, splenohepatomegaly, cutaneous petechiae, bone defects, hypochromic anemia, and fever. The bone defects usually are multiple and have the same appearance as that in the other two types of the disease. Histologically the lesions, which occur both in the viscera and the skeletal bones, contain histio-

cytes, giant cells, and eosinophiles. But there are no cholesterol crystals in the tissue spaces and no foam cells. Thus, this disease differs both clinically and pathologically from that described by us in our present series.

2. Eosinophilic granuloma. These lesions may be multiple but usually have one main site, and are found in children, adolescents, or in young adults. They tend to run a chronic benign course. The skull, especially the frontal bones most often are affected. In about one-quarter of the patients, injury at the site precedes the symptoms and signs. The patient's general health usually is good. In a few instances a mild eosinophilia is found. Thus, the history, X-ray, and clinical findings do not differ greatly from our present cases.

Microscopically the lesions are composed of histiocytes which contain much cellular debris and blood pigment, but usually no lipoid. They contain scattered multinucleated giant cells, with varying numbers of eosinophiles. No extracellular cholesterol crystals are found, and there is very little fibrous connecting tissue. In some of these characteristics they differ from our cases. In 1946 Wheeler[15] described a characteristic lesion of this type in a young man of 34 years. His case was similar to ones we have described, except for the histologic differences.

3. Schüller-Christian's disease. This is usually considered to be a disease of childhood. It is marked by widely disseminated visceral and skeletal lesions of a granulomatous nature. The classical triad of Schüller-Christian's disease (granulomas, hypercholesterolemia, diabetes insipidus) may be completely or only partially present. The disease may occur in adults, even as late as in the fifth decade. Cases with solitary lesions have been reported. As indicated above it has been suggested by some that these tumors originally may exist as eosinophilic granulomas. However, this has not been proven. The microscopic characteristics of the lesions in the viscera and skeletal bones are similar and have been described by many. They contain a large number of lipoid-containing histiocytes (xanthoma or foam cells). Many of these histiocytes contain blood granules or cell debris. The foam cells tend to be arranged around blood vessels. There are numerous multinucleated giant cells and, in places, lipoid crystals. Here and there a small amount of infiltration with few eosinophiles can be seen. In the younger lesions, xanthoma cells in a vascular matrix is the predominant feature, while in the older lesions there are various stages of fatty degeneration, foreign-body reaction, and fibrosis.

Hypercholesterolemia is not necessarily present, as we now know. In some cases the serum cholesterol may be elevated owing, it is thought, to the sudden and intermittent release of fatty substances into the blood stream. In 1941, Dandy[16] described, in a larger series, a small group of five cases which he thought represented Schüller-Christian's disease. His first case was that of a 46-year-old man who had noted a slight swelling of the left upper eyelid for a little over two months. In this man the lesion clinically and pathologically was not unlike mine, and indeed may belong to the same group. However, this is not true of the other four patients, as there were no skeletal or skull defects.

Certainly our three patients do not seem to have had a dermoid or epidermoid cyst, or a giant-cell tumor. The question then arises as whether their lesion is a diploic hematoma or some form of histiocytic granuloma. The relatively infrequent history of trauma in our cases would seem to rule out the chance of hematoma. The characteristic feature of all the 15 patients described here is the presence of masses of cholesterol crystals. Hence, Hanbery and Rayport[1] have suggested the name of cholesterol granuloma. One may speculate that these lesions are a chronic or benign form of the histiocytic granuloma of Schüller-Christian's disease occurring in adults as a solitary lesion with a tendency to be localized in the fronto-

TABLE 1

SUMMARY OF CLINICAL CHARACTERISTICS OF 15 PATIENTS WITH CHOLESTEROL-CONTAINING GRANULOMA

1. Presenting symptoms:	Proptosis and diplopia	All patients
	Failing vision	5 patients
2. Duration of symptoms:	Few weeks to many years	
3. History of trauma:		5 patients
4. Age:		28–63 years
5. Sex:		13 male, 2 female
6. Side affected:		7 right, 8 left
7. Proptosis and limitation of upward and outward gaze:		All
8. Blurred vision and papillitis:		4 patients
9. Hypercholesterolemia (4 patients only tested):		1 patient
10. Wassermann, positive:		1 patient
11. Recurrence after surgical removal:		None

orbital region, and producing proptosis. Reese[17] in discussing these tumors favors this point of view and refers to them as solitary histiocytomas. It is possible, though far from proven, that these lesions may have originated as esinophilic granulomas, which then may have undergone gradual fatty degeneration and fibrosis. If this be true, our cases will be brought into relation with that of Wheeler[15] on the one hand, and with Dandy's[16] case on the other, interconnecting the whole as one group. Thus, the present series, all of which contain lipoid material and fibrous tissue with few eosinophiles, may represent the most chronic form of the granulomatous lesion.

No matter how one may wish to categorize these lesions, the matter of practical importance is that they are essentially benign. The pathologic process is that of a granuloma and not a neoplasm; it is not one of the lipoidoses which are based on faulty cholesterol metabolism; when seen as a solitary lesion in adults there is little likelihood that other lesions will appear; and, finally, local excision leads to rapid and permanent relief of symptoms.

SUMMARY AND CONCLUSIONS

1. Three similar case reports of cholesterol-containing granulomas of the fronto-orbital bones resulting in unilateral proptosis are presented.

2. Twelve other cases reported in the literature are discussed.

3. The lesion in each case was solitary and occurred in young or middle-aged adult patients, only two of whom were female; it had been in existence for many years in some cases.

4. Each patient presented the signs and symptoms of unilateral proptosis and diplopia.

5. In four cases a definite history, and in one a suggestive history, of trauma was found.

6. Radiologically each patient presented an osteolytic lesion of the lateral portion of the supraorbital ridge; seven were in the right orbit and eight in the left.

7. At operation, in all patients a cavity of the bone was found which was filled with soft yellow-gray, amorphous, gritty material. No evidence of invasion could be found.

8. In each case the microscopic appearance of the tissue removed was similar, showing many large masses of cholesterol crystal clefts, foreign-body giant cells, foam cells, histiocytes, inflammatory cells, blood pigment, and fibrosis.

9. Local removal resulted in uneventful recovery with no recurrence.

10. A conservative operative approach is recommended.

11. The etiology and pathogensis of these lesions is unknown, so they must be classified merely as granulomas. It is speculated that these lesions may be related to the histiocystic granuloma of the Schüller-Christian type on the one hand, and to

eosinophilic granuloma on the other.

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RETINAL DETACHMENT SURGERY*

A UNIVERSITY HOSPITAL SERIES COMPARED WITH A PRIVATE PRACTICE SERIES

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A dramatic change in the prognosis of eye disease has occurred in the field of retinal detachment, a condition which only 25 years ago was all but synonymous with blindness. Prior to 1930, when Gonin's[1] operation first began to be widely performed, the therapeutic approach to retinal detachment was crude and ineffective. In the mid-19th century Graefe[2] had advocated multiple discission of the retina, and somewhat later deWecker[3] and Deutschmann[3] proposed such procedures as dividing vitreous strands and tearing the retina.

A number of abortive attempts to effect chorioretinal adhesions were made very early. Fano[3] tried with iodine in 1863, deWecker[3] with the actual cautery in 1884, and Abadie[3] with the galvanocautery in 1893. It was in 1870 that deWecker[3] advanced the

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theory that retinal detachment resulted from rupture of the retina, permitting a flow of vitreous into the chorioretinal space, and in 1882 Leber[4] introduced the vitreous traction theory.

It remained for Gonin, many years later, to be sufficiently convinced of the soundness of these pathogenetic theories to attempt by radical means to seal off the tears. As early as 1919[5] he began to talk about thermopuncture, and in the 1920s,[6-9] he wrote a series of papers on the subject. He reported that the rate of cure was 60 percent during this period but his work was not widely understood by the English-speaking world until he described it in 1929[1] before the International Congress of Ophthalmology at Amsterdam. Since that time Gonin's theory and general approach have been widely applied. The techniques employed have been gradually improved and, in 25 years, the favorable prognosis for retinal detachment surgery has risen from virtually nil to better than 80 percent in some recent series.

Scleral resection as a supplementary approach to the repair of retinal detachment was introduced as early as 1903 by Müller.[10] Since massive vitreous hemorrhages and vitreous loss often resulted, however, this procedure was reserved for desperate cases and performed only by the boldest surgeons. Cures were achieved by such pioneers as Blaskovics,[11] Torok,[12] Elschnig,[13] and others,[14-20] but the operation was generally regarded as too dangerous to attempt until Lindner began using it in 1933. Since then the technique has been improved by Pischel,[21,22] Borley,[23] Lindner,[24] Shapland,[25] and others,[26] and scleral resection is now commonly used in complicated cases.

In the following report the results of retinal detachment surgery performed at the University of California Hospital since the first operation in 1917 are reviewed and compared with the results obtained in a larger series performed in a three and one-half year period (1951-1954) in connection with a private practice.

University of California Hospital series

Operations performed 1917-1929

In this era only three retinal detachment operations were performed at the University of California Hospital. Dr. Walter Scott Franklin's operative note on the first of these (December 21, 1917) is of interest:

> Local anesthetic, 4% cocaine. The needle was forced through the sclera on the outer side of the eye and a current of a single-cell dry battery allowed to flow a few seconds. The needle was then reinserted in another area about ½ inch from the first location and the current again allowed to flow for a few seconds. The eye was dressed with yellow oxide of mercury, H. R.

The patient was discharged the following day, his condition unimproved. Dr. Franklin was apparently not impressed with the available surgical methods of the time since in 1920 he treated a case of retinal detachment ". . . by general elimination measures, mercury rubs, and with local atropine and hot compresses to the eye." This patient was discharged after one week, the retina still detached and the vision unimproved.

In 1926, Dr. Joseph Crawford performed a posterior sclerotomy. This was the first successful detachment operation in the records of the University of California Hospital. The following year Dr. Warren Horner repeated this procedure in another case but was unsuccessful. In this same year a third case was treated with "sweats and sedatives."

Operations performed 1930-1939

From 1930 to 1939, inclusive, 11 Safar diathermy operations* were performed. In this procedure penetrating Walker pins were inserted in the sclera and an extensive exudative choroiditis was produced. As many as 30 pins were used and usually a considerable amount of subretinal fluid escaped. The pro-

* Dr. Pischel, upon his return from Vienna at this time, demonstrated at the University of California Hospital a new modification of the Gonin operation which had been developed in Europe by Safar.

cedure was successful in four instances and failed in six; the final results in the 11th case were not recorded.

In 1932, electrolysis was combined with posterior sclerotomy in four operations performed by Dr. Joseph McCool. The electrolysis was applied to the exposed sclera over the area of the retinal hole and in several instances was combined with a number of small scleral trephinings. One case was a clear-cut success; the results in the other three were equivocal.

These early cases are of more historic than practical interest and have been omitted from the statistical data that follow.

Operations performed 1940-1954

During this 15-year period, 123 cases were operated upon for retinal detachment by attending staff ophthalmologists and residents in accordance with Gonin's approach to the therapeutic problem. The end-result was recorded as a cure or failure only after a postoperative period of two months or more. After the first procedure, 24 of the failures were reoperated one or more times. In the final analysis a permanently attached retina with useful vision was considered a cure, a still unattached retina a failure.

In general the procedure recommended by Pischel was observed, as follows:

Preoperative management. (1) Repeated fundus examinations were made in attempts to localize the retinal tear or tears, and a detailed drawing of the fundus was constructed. (2) The patient was placed at bed rest with binocular bandages for approximately five days.

Surgery. (1) By direct ophthalmoscopic examination each tear was localized and the corresponding site on the sclera marked. (2) The area around the site was then coagulated with the Walker diathermy machine, and subretinal fluid was drained by means of Pischel pins, or a Wheeler knife if necessary. (3) In certain complicated cases the diathermy operation was supplemented by scleral resection.

Postoperative management. (1) The patient was kept at bedrest with binocular bandages for approximately 14 days. (2) Pinhole glasses were prescribed for wear during a six-week convalescent period at home. (3) Fundus examinations were made in the office every 10 days during convalescence.

Results. The final results from 20 of the 123 operations were not recorded. Of the remaining 103 in which the records were complete, 64 percent resulted in cure and 36 percent in failure. This represents the combined average of the cures in the group of private patients treated at University of California Hospital (70 percent) and the group of clinic patients (58 percent) treated at the same institution. In 14 of the 103 cases the diathermy operations were supplemented by full-thickness or lamellar scleral resections. Only four of these resulted in cure but three of the four were among the last six cases in the series, all of which had been subjected to lamellar scleral resection as a primary procedure.

The records of the 103 cases in which the final results of operation were known were examined for any findings or factors which might have had a bearing on the results of operation. The data in Table 1 are of interest.

Contrary to the generally accepted view that retinal detachment complicated by myopia decreases the chances of achieving reattachment of the retina, the rate of cure in cases with myopia was as high as it was in the group without myopia.

The value of finding the retinal tear preoperatively was amply demonstrated. In 87 cases the tear was found and 60, or 69 percent, of these were cured by operation; in the 16 in which the tear was not found, the cure rate was only 37.5 percent.

Of prognostic value was the anticipated finding that a low rate of cure obtained in cases of total detachment, aphakic detachment, and cases requiring reoperation. In this connection, however, it should be stressed

TABLE 1

RESULTS IN THE UNIVERSITY OF CALIFORNIA SERIES

Total Number of Cases with Final Results Known	103	Cures 66 (64%)	Failures 37 (36%)
Myopia (5.0 D. or more)	14	8	6
Retinal tear found	87	60	27
Retinal tear not found	16	6	10
Total retinal detachment	12	3	9
Patient reoperated	22	5	17
Aphakia	13	5	8

that unsuccessful procedures should not prejudice additional surgical attempts when any possibility of a cure remains. Two patients in this series were saved from total blindness by a fifth procedure.

The proportion of males to females (65:58) was statistically insignificant. There was insufficient information on the relation of trauma to retinal detachment, and on the relation of the drainage of subretinal fluid during operation to cure. With regard to this latter point, however, the review of the surgical records conveyed the unmistakable impression that failure to obtain adequate drainage greatly decreased the changes of success.

CASES OF SPECIAL INTEREST

In two cases of this series, detachments of long duration were cured in young patients. Their histories follow:

Case 1. A 15-year-old white boy was first examined in 1950. He related that the vision of his right eye had suddenly decreased eight months before. Examination revealed myopia of eight diopters in both eyes and a large inferior detachment and large retinal hole in the right eye. There was no history of trauma. The diathermy operation was performed and an excellent result obtained. Four years later this patient had a normal peripheral field with a central cecal scotoma, and a corrected visual acuity of 20/80.

Case 2. A 14-year-old white girl had been hit on the head with a baseball 11 months prior to her first visit to the University of California Eye Clinic in April, 1953. She complained that immediate and marked loss of vision in her right eye had occurred after the injury and that the vision in this eye had deteriorated progressively. On first examination a total retinal detachment and a large retinal cyst were disclosed. There was no apparent light perception and the condition was regarded as hopeless. Four months later, however, another examiner thought he detected some light perception. The diathermy operation was performed and the cyst drained. No retinal tear was seen before or during the operation. The retina was still flat 18 months postoperatively. The field was slightly constricted and the patient had a corrected visual acuity of 15/200.

These cases demonstrate the influence of age on prognosis. The results of efforts to repair long-standing retinal detachments in elderly patients have been uniformly poor.

PRIVATE PRACTICE SERIES

Through the courtesy and kindness of a leading private practitioner and his associate we have been permitted to include in this report a brief summary of all cases of retinal detachment treated surgically in the course of their private practice from August, 1951, through December, 1954. In this period of three and one-half years, 376 eyes were operated upon by means of 294 diathermy procedures, plus 224 diathermy procedures combined with lamellar or penetrating scleral resections. Altogether 518 procedures were performed on the 376 eyes (an average of 1.7 per eye), scleral resection having been combined with diathermy in 43 percent of all operations.

The results are shown in Table 2.

TABLE 2

RESULTS IN THE PRIVATE PRACTICE SERIES

	No. Cases	Cures	Failures	% Cures
New Cases (all surgery performed in private practice series)				
(1) One procedure	224	202	22	
(2) Two or more procedures	89	52	37	
TOTAL	313	254	59	81
Old Cases (previous unsuccessful) surgery performed elsewhere)				
(1) One procedure	40	21	19	
(2) Two or more procedures	23	6	17	
Total	63	27	36	43
TOTAL OF ALL CASES	376	281	95	75

Of the total of 376 cases, 75 percent were cured. If the 63 old cases are excluded, that is, the early one-sixth of the series which were referred after at least one unsuccessful operation elsewhere, the rate of cure in the remaining 313 cases was 81 percent.

DISCUSSION

It was surprising to find that in a teaching center the size of the University of California Hospital, only 123 cases of retinal detachment were subjected to surgical treatment in a 15-year period. One determining factor may be that Dr. Pischel, who has been an outstanding leader in the field of retinal detachment surgery since 1930, practices in the same city and uses other hospital facilities. The cure rate of 64 percent in the University of California series is perhaps better than might be expected in a teaching hospital which tends to attract cases in which previous surgery has been successful or in which the prognosis has been poor from the start.

By virtue of the pioneering at Stanford Hospital by Pischel and Borley, scleral resection was used rather early at the University of California Hospital. The first full-thickness resection was performed here in 1943. In the early period of its use the procedure was undertaken for the most part only in seemingly hopeless cases. Lamellar scleral resection as it is performed today is a relatively safe and simple technique, however, and is steadily coming into wider use. Indicative of its increasing popularity is the fact that of the last eight operations for retinal detachment performed at the University of California Hospital, scleral resection was the primary procedure in six and a secondary procedure in one of the other two. The indications for its use that are followed at this hospital are those laid down by Lindner,[24] Shapland,[25] Berliner,[26] and Pischel and Kronfeld.[21, 22]

In an effort to uncover the reasons for the disparity between the results at the University of California Hospital and the private practice series, the details of the preoperative, operative, and postoperative management of the latter series were examined and may be summarized as follows:

The treatment of any case begins with an exhaustive ophthalmoscopic examination. Not less than an hour is spent observing the smallest possible details, including any obscure holes. Nearly all of this examination is by direct observation with a giant electric ophthalmoscope, supplemented by observation with a binocular indirect ophthalmoscope. A large, full-page diagram of the fundus is constructed and each vessel is traced as far out as possible. In this way no part of the retina is overlooked and no detectable

retinal hole is missed. A thorough examination of the vitreous is also made with the slit-lamp and Hruby lens.[21]

The patient is then put at rest for approximately five days so that the effect of any settling of the retina may be observed. In some instances this rest period is prolonged if it seems that more settling may thus be effected. Further detailed and time-consuming examination of the retina is undertaken during this period since holes not previously seen may become demonstrable. Thorough familiarity with the particular fundus in question is regarded as of first importance to facilitate orientation at the time of surgery when conditions are never favorable for the best funduscopy.

This preoperative evaluation of the problem and the decision as to choice of procedure seem to be by far the most important factors in determining the end result. The actual techniques of operation, whether diathermy alone or diathermy combined with scleral resection, are not difficult to perform once a few basic principles and methods have been learned.

The most important single necessity at the time of surgery is the precise localization of the tears. This is painstakingly accomplished by direct observation, usually as soon as the sclera has been bared. The assistant marks the sclera with methylene blue. The diathermy needle is then applied to this area and the localization rechecked with the ophthalmoscope. The next most important step is to secure adequate drainage, by means of Pischel pins or a knife if necessary. Again the careful preoperative evaluation will have indicated where drainage attempts are apt to be most fruitful.

From a review of the case histories and representative surgery of the two series reported herewith, it may be concluded that attention to minute detail, regardless of the time consumed, and the taking of infinite pains at every step, but particularly preoperatively in preparation for surgery, account for the markedly higher incidence of success in the private practice series. At the risk of redundance, these observations are offered in the belief that an observer may sometimes recognize, and justifiably emphasize, a reason for success which the surgeon himself takes for granted.

Summary and Conclusions

In the course of this study all cases of surgically treated retinal detachment in the files of the University of California Hospital were reviewed. The operations performed before 1940 are described chiefly for their historic interest. In the period 1940-1954, 123 cases were subjected to diathermy and, in 14 cases, also to scleral resections, in accordance with the principles of Gonin and the procedural recommendations of Pischel. In the 103 cases in which the final results were recorded, cures were achieved in 64 percent. After an initial failure, 24 patients underwent one or more additional operations and at least five of these were cured. Two patients were saved from total blindness by a fifth procedure. At this hospital, lamellar scleral resection is rapidly gaining favor as an initial procedure in complicated cases.

Two cases are presented in which cures for retinal detachment of long duration were effected in young patients.

For purposes of comparison a series of retinal detachment operations performed by two ophthalmologists prominent in the field of retinal detachment is presented. In 376 consecutive cases treated surgically during a three and one-half-year period, 75 percent were cured as a result of 518 procedures, or an average of 1.7 procedures per patient. If the 63 cases that had undergone previous unsuccessful surgery elsewhere were eliminated, the rate of cure was 81 percent.

From a study of the preoperative, operative, and postoperative management in the two series, it was concluded that painstaking attention to minute detail, particularly preoperatively, was largely responsible for the markedly higher incidence of success in the private practice series.

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TOTAL DETACHMENT AND REATTACHMENT OF THE RETINA*

In herpes zoster ophthalmicus

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Detachment of the retina is not a frequent complication of herpes zoster ophthalmicus.[1] Edgerton[2] mentions only one case in his exhaustive review in 1945, that reported by Scott[3] in the British literature of 1932. The present paper describes three further cases in which the onset of herpes zoster was followed by retinal detachment.

The first case, seen by H. L., was a complete funnel detachment. The second case, seen by G. W., was a partial detachment. The third case, seen by H. R., was a partial detachment with a peripheral hole in the superior nasal retina.

The question arises immediately as to whether the detachments were incidental to the herpes or resultant therefrom. In the first two cases, severe iridocyclitis was present and spontaneous reattachment occurred without an operative procedure. In neither case was a hole found. These cases suggest that an exudative choroiditis was the mechanism of detachment. The third case also showed iridocyclitis but differed from the first two in that a hole was found and repaired. Reattachment followed repair of the

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hole; however, the gradual character of the reattachment suggested that here, too, an exudative choroiditis was operative in the detachment.

It is thought that the mechanism of detachment was a lifting of the retina by an extravasation from the choroid, secondary to a histamine reaction in the choriocapillaris. This might be caused either by direct infection of the choroid by the herpes zoster virus, or by antidromic impulses from an infected gasserian ganglion[4]

In this connection it is interesting to consider the known pathology of herpetic skin lesions. In the skin, a tense vesicle is formed by a collection of serous exudate beneath a firmly bound tissue. Eventually the vesicle ruptures and secondary infection occurs, producing the weeping lesion typical of the disease. Beneath the retina, an exudate from the choroid would not be contained as beneath the skin, but would be free to spread with little resistance from the ora to the disc; thus, instead of localized vesicles, a diffuse elevation, that is, a detachment, would result.

CASE 1

The case of H. L. was that of a 40-year-old woman purse inspector, who was admitted to the medical service on May 19, 1952, with a typical history of acute herpes zoster. Three days previously she had suffered a chill and headache, followed in a few hours by a rash on the face. Upon admission she was in acute distress with high fever and massive edema of the face. Both eyes were closed by the edema. There were numerous small vesicles over the entire body. The diagnosis was herpes zoster generalitivus.

The next morning the patient was seen by the eye service. The vesicles were noted to be chiefly on the right side of the face along the course of the ophthalmic division of the fifth nerve. Vesicles extended to the tip of the nose. The cornea and the conjunctiva were grossly edematous, and the globe deeply injected. The pupil was static and very small. The fundus could not be seen, due to the corneal edema and the constricted pupil. To the medical diagnosis was added: herpes zoster ophthalmicus (1) keratitis, (2) iridocyclitis.

Local treatment was started and consisted of cortisone solution (0.5-percent every two hours), aureomycin ointment (three times daily), and atropine solution (one percent, twice daily). On this therapy the pupil dilated to five mm. and the patient was more comfortable. By June 2nd, 12 days after admission, the cornea had cleared somewhat, although it was still thickened and had folds in Descemet's membrane. Slitlamp examination of the anterior chamber showed considerable precipitation of pigment on the cornea and lens, indicative of the severity of the iridocyclitis which by now had been largely suppressed by steroid therapy.

The patient was observed daily, and the skin and anterior segment were noted to improve steadily. No attempt was made to examine the fundus until the patient reported that she had no vision in the right eye. Testing confirmed this; there was not even light perception.

Examination with a five-volt ophthalmoscope revealed a funnel-shaped detachment with the retina lying against the lens. To the previous diagnosis was added massive detachment of the retina, secondary to an exudative choroiditis. Direct treatment of the detachment was not considered feasible at this time. Atropine and topical cortisone were continued, in an attempt to quiet further the diseased eye and to reduce the photophobia in the uninvolved eye of which the patient complained. At one point subconjunctival cortisone was started, but this was abandoned because of difficulty in administering the injection.

When the patient was discharged to the out-patient service, after 27 hospital days, the right eye was still moderately injected and without light perception, and photophobia was still present.

During the next six months, the patient was seen regularly and frequently and

showed a gradual lessening of the ciliary injection. Treatment with cortisone, four times daily, and atropine (one percent, three times daily) was continued. In May, 1953, the patient stated that she could see shadows. Visual testing confirmed that light perception had returned in the right eye. Ophthalmoscopic examination revealed that the retina was no longer against the lens, but a very cloudy media prevented any visualization of the retina itself.

In October, 1953, the media, although still hazy, had cleared sufficiently so that the retina could be seen. It seemed to be flatly detached above, but largely in place inferiorly. In February, 1954, this opinion was confirmed through a greatly cleared media and the patient counted fingers at one foot. A field examination with a bright light at this time showed an irregular 15-degree field with poor fixation.

Through the remainder of the year the vitreous continued to clear, until, with the exception of a few small floaters, it was entirely transparent. Vision improved to finger counting at five feet, and the field steadily enlarged, until a nearly full peripheral field for light was obtained in February, 1955, (fig. 1). Central fields showed an absolute central scotoma of four degrees.

On ophthalmoscopic examination the retina was now seen to be completely attached. The areas of relative scotoma medially and laterally were due to areas of retinal degeneration that were clearly defined by pigment deposits. The macular area also showed degeneration. It had evidently been out of contact with the choroid too long to survive.

Case 2

The case of G. W. was that of 58-year-old Negro, who had suffered a severe attack of herpes zoster ophthalmicus on the right side in September, 1950. There was an associated paresis of the third and fourth cranial nerves, punctate keratitis, iridocyclitis, and secondary glaucoma. In October an iridencleisis was done and the intraocular pressure reduced.

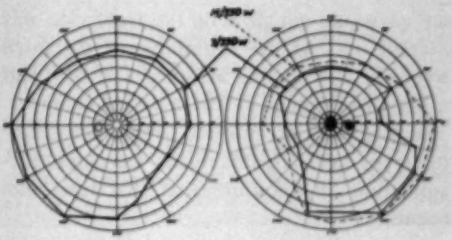

Fig. 1 (Lincoff, Wise, and Romaine). *Case 1.* Visual fields in February, 1955, almost three years after the attack of herpes zoster ophthalmicus.

G. W. first saw the case in December, 1950. At that time vision was 20/200 in the involved eye. In addition to the complications already noted, a large inferior bullous detachment was observed. No hole could be found. Intraocular pressure measured 37 mm. Hg in the right eye and 19 mm. Hg in the left. Pilocarpine (two percent, three times daily) controlled the elevated pressure in the right eye, and was continued throughout the following year. This was the only medication used. No treatment of the detachment was attempted.

By February, 1952, 14 months after the detachment was first noted, and 18 months after the onset of the herpes zoster ophthalmicus, the retina had spontaneously reattached and vision of the right eye was 20/30. When last seen in February 1953, the retina was noted still to be completely attached, and vision in the right eye was 20/20.

Case 3

The case of H. R. was that of a 58-year-old man who developed herpes zoster ophthalmicus in the right eye in July, 1950, with typical skin lesions along the course of the first division of the trigeminal nerve. During the first two weeks the nasociliary branch was considered not to be affected. Vision was unimpaired, and examination of the anterior and posterior segments of the globe was negative, as was a field examination. Due to the severe pain, X-ray radiation of

the gasserian ganglion on the right side was carried out. At the beginning of the third week the eye became inflamed and cells and a flare were noted in the anterior chamber. The diagnosis of anterior uveitis secondary to herpes zoster ophthalmicus was made. Examination of the fundus at that time was negative.

One week later the patient complained of a flashing light and a curtain in the lower nasal field. Ophthalmoscopic examination now showed a large bullous detachment in the superotemporal area, with a hole in the far periphery. A surgical procedure was carried out to seal the hole; however, the retina did not reattach immediately after this procedure but reattached very gradually over several months, after which normal visual acuity was restored.

Summary

Three cases of retinal detachment as a complication of herpes zoster ophthalmicus are presented. All were characterized by the presence of iridocyclitis, bullous detachment, and the tendency for spontaneous reattachment. In only one case was a hole found and treated. The mechanism of detachment is thought to be an exudative choroiditis.

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A COMPARATIVE STUDY OF GONIOSCOPIC METHODS*

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I. Introduction

Opinions vary as to the best method of gonioscopy. Each variety of equipment has its own particular merit, its specific disadvantages, and, needless to say, its own ardent proponents. The purpose of this paper is to discuss the methods for gonioscopy which are now available, and briefly to describe the optical principles involved. We hope that this may help the ophthalmologist to choose the equipment which may best fulfill his individual requirements, within the limits of his resources and facilities.

The essence of satisfactory gonioscopy is the obtaining of a highly magnified and brilliantly illuminated view of the anterior chamber angle. For reasons which will be presently considered, the first requirement is a contact lens. This should be capable of easy insertion, and should stay properly positioned without the need of additional support. There should be no discomfort to the patient during the examination, and the lens itself should produce no mechanical injury to the globe. The lens should be of perfect optical quality to minimize distortion, should be durable, and, in addition, should be capable of being

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easily cleaned and thoroughly sterilized.

A magnifying device of some type is essential so that the image of the angle transmitted by the contact lens may be enlarged to such a degree that the details of the angle structure can easily be studied. This magnification should be capable of variation, depending on the requirements of the individual case. Steady support of the magnifier is necessary, particularly in the larger magnifications where the distance from the observer to the eye being examined becomes more critical, and the field of vision becomes smaller. Binocular observation, with the advantage of stereopsis, is also important in most cases.

The third essential portion of the gonioscopic system is the illuminating device. The light afforded by it should be of high intensity and capable of sharp focus. It should normally be directed coaxially with the observation system, yet should be capable of a degree of divergence in one or the other direction. Should the situation warrant, it should be capable of use as a retro-illuminating device so that the chamber angle may be observed by indirect illumination. The majority of observers prefer a slit-type beam of variable width and angle to study the contour of the chamber angle.

The various types of devices available for each of these three units of the gonioscopic optical system will now be discussed, together with the advantages and disadvantages of each.

II. Contact lenses

A. Theoretical considerations

Before considering the various types of contact lenses available for the study of the anterior chamber angle, we should first consider why artificial means are necessary for its observation. Direct observation of the angle from directly in front of the eye is, of course, impossible because of the opacity of the overlying corneoscleral limbus. It therefore becomes necessary, by means of oblique paths of light, to study the opposite angle. This is ordinarily impossible because of the total reflection which occurs at the cornea-air interface.

One of the fundamental principles of geometric optics is expressed in Snell's law, which states that the ratio of the sine of the angle of incidence to the sine of the angle of refraction at a given interface between two substances of varying optical densities, is inversely proportional to the indices of refraction of the two materials. From this it follows that the sine of the angle of refraction, and therefore the angle of refraction itself, is always greater on the side of least optical density.

The maximum angle of refraction can only be 90 degrees, for at this point the emergent ray is parallel to the interface, or its tangent at the point of emergence. The angle of incidence in the case of decreasing densities is somewhat less than 90 degrees. Should the angle of incidence be somewhat more than that which would produce an angle of refraction of 90 degrees, the beam is reflected back into the original medium and no transmission occurs. The minimum angle of incidence at which total reflection occurs is termed the critical angle and can only occur where the first medium is of greater index of refraction than the second.

The critical angle becomes of considerable importance in gonioscopy because it is this which, in most cases, prevents the observation of the anterior chamber angle. The critical angle of the cornea-to-air interface, or, more properly, the tear film-to-air interface, is about 49 degrees. Total reflection does not occur at the aqueous-cornea interface because of the slightly lower index of refraction of the former.

Assuming the radius of the curvature of the cornea to be roughly 7.8 mm., the majority of light rays projected from the iris root will strike the cornea-air interface at an angle greater than the critical angle and total reflection will occur. Direct, unaided observation is therefore not feasible except in isolated instances.

This difficulty could theoretically be overcome in one of two ways. Were the index of refraction of the cornea and aqueous less, total reflection would be less of a problem and observation would be improved. This is manifestly impossible of attainment. On the other hand, if the rays emanating from the anterior chamber angle could be made to strike the air interface less obliquely, their transmission would be enhanced. In the case of the Koeppe and Troncoso contact lenses this is accomplished by substituting for the corneal surface a glass or plastic surface of a different radius of curvature. On the other hand, the emergent beam may be deflected by a mirror incorporated within the lens, or by a totally reflecting surface, which will so change its course as to overcome the critical angle at the lens-air interface. For the purposes of classification we have chosen to designate the former as "direct-vision" lenses and the latter as "indirect-vision" lenses. The purpose of each is obviously identical.

B. Direct-vision lenses

To Trantas[1] must be given credit for the first observations of the anterior chamber angle. He made use of direct and indirect ophthalmoscopy for its study, without the use of a contact lens. He was able to see the nasal and temporal angles in 25 percent of his cases. In those with particularly shallow chambers he made use of digital pressure on the globe to alter its curvature.

Subsequently Salzmann[2] made use of the original Fick keratoconus contact lens for direct examination of the iris angle, following unsuccessful attempts with unaided ophthalmoscopy. He later devised his own lens and filled the space between it and the cornea with normal saline. Koeppe,[3] in 1919, devised a somewhat thicker lens, the so-called Koeppe "A" lens, which overcame some of the astigmatic distortion encountered by Salzmann. This lens possessed a groove about its base so that it could be held in place by the lids, and also had a small indentation at its vertex. Koeppe's observations were made at the slitlamp with the patient in an erect position, and this indentation received a knot in the head bandage which also helped to hold the lens in position. In 1925, at the suggestion of Troncoso,[4] the Koeppe "C" lens was devised, which had an increased outer radius of curvature and did not possess the indentation at its vertex. The inner radius of curvature remained the same as that of the cornea, so that the second lens was considerably thicker and heavier than the original.

As now manufactured the Koeppe lens is made of barium crown glass having an index of refraction of 1.516. Its outer radius of curvature is 12.5 mm., and the inner is 7.5 mm. It produces a magnification of about ×1.5 and is available in diameters of 16, 18, and 20 mm. to fit varying palpebral fissures.* Numerous variations of the Koeppe lens have been proposed since its inception. In 1940, Barkan[5] described a molded lucite lens similar in appearance to the glass Koeppe lens but possessing a detachable handle to facilitate positioning. In 1941, Friedman[6] proposed a contact lens with side-by-side perforations two mm. apart so that fluid could be injected through one and air ejected from the other.

Further variations of the Koeppe lens were proposed to facilitate its use in the goniotomy operation. Ellis[7] described a lucite lens which was edged to promote ease in manipulation, but possessed the handle of the previous Barkan lens. In 1950, Barkan[8] described the glass goniotomy lens currently in use. This does not possess a handle but instead has two small depressions on its outer surface to aid in positioning. It is manufactured in two sizes having diameters of 11 and 13 mm., and possesses a magnification of ×1.4. Its radii of curvature are substantially that of the present diagnostic contact lens.

In 1945, Troncoso[9] proposed a direct-vision contact glass of entirely different de-

* These lenses are manufactured by Parsons' Optical Laboratories, San Francisco, California.

sign from the previously existing Koeppe lens. This was fashioned of acrylic plastic having an index of refraction of 1.48. It did not possess a groove around its base as did the Koeppe lens, but rather was designed with a wide scleral shoulder over which the lids could ride in order to maintain it in position. As presently manufactured, its outer radius of curvature is 8.5 mm .and its magnifying power is therefore greater than the Koeppe lens, being about two diameters. This lens was further modified by Troncoso[10] in 1951 to provide for a small tube at its vertex through which fluid could be injected to eliminate air bubbles.

At this point we should consider the relative advantages of plastic and glass contact lenses. Plastic lenses are light in weight but are softer and more easily scratched than the glass variety. Neither can they be as highly polished. In addition, they possess poor wetting qualities and for this reason air bubbles commonly cling to their surfaces and interfere with good visibility. The use of various wetting agents has been advocated to overcome this difficulty. Glass lenses, on the other hand, wet easily and, being harder, their surfaces are most resistant to abrasion. They possess the disadvantage, however, of being considerably heavier in weight and more easily broken. An additional disadvantage of the plastic lens described by Troncoso is that it is easily smeared by the oily meibomian secretions if the lids brush over it.

The advantages of the direct-vision lens are several. In the first place, binocularity, and therefore stereopsis, is easy to maintain even with considerable movement of the magnifying device. This permits examination of a large portion of the angle from one position of the observer. Because of the supine position of the patient, iridodonesis of small degree becomes readily visible. Capsular exfoliation on the posterior surface of the iris can also be observed. The patient himself is more comfortable with this lens than with the indirect types. A greater total magnification is also possible with these lenses because of the basic magnification produced by the lens itself due to the curvature of its outer surface. Also, it is our opinion that more precise evaluation of the narrowness of the anterior chamber angle seems possible than with the indirect methods which will presently be described.

There are also certain disadvantages which are inherent in the use of the direct-vision type of lens. A good light source, a microscope complete with support, as well as space for a table or reclining chair, are all necessary adjuncts. If the microscope from the office slitlamp is used, the nuisance of removing it may well deter the user from the performance of frequent gonioscopic examination.

Another disadvantage in the use of this type of lens is that the observer must move in a 360-degree arc around the patient. This, of course, necessitates additional space in the examining room. Also, if the patient possesses a prominent nose, the upper temporal quadrant may be difficult to visualize. A certain amount of astigmatic distortion is to be expected because of the obliquity of the line of vision through the lens, although this varies greatly, depending upon the position of the observer. Prismatic deviation of the image also occurs, but presents no particular difficulties. The solution filling the lens can be displaced by air, spoiling visibility, and an assistant is often needed to help hold the lens in position.

C. Indirect-vision lenses

In order to enable the observer to use the illuminating and magnifying systems of an already existing slitlamp to examine the iris angle, Goldmann,[11] in 1938, proposed the use of a contact lens of different design. This lens contains a mirror which reflects the slitlamp beam into the angle and also reflects emergent rays into a direction almost parallel with the optic axis and into the microscope. The anterior surface is flat and the posterior surface is rounded to conform to the shape of the cornea. There is a scleral shoulder to

enable it to be held in contact with the globe by the lids. Since the patient is in the upright position it is made as light as possible; it is fashioned of plastic and has a total weight of 4.2 gm.

The lens is used with the patient in the upright position, and requires a solution to fill the space between lens and cornea. It is a most awkward lens to use and bubbles are frequently a source of annoyance. Nevertheless, a good view of the angle can be obtained, though there is difficulty in maintaining binocularity in the horizontal meridian. According to Donaldson[12] it is the best lens to use in taking stereoscopic pictures of the angle.

An improvement in this type of lens was described by Allen and O'Brien[13] in 1945. Their lens is similar in principle to the Goldmann lens except that the reflecting surface becomes one side of the lens, its angle being such as to make it a type of totally reflecting prism. The anterior surface is flat and at right angles to the emergent rays. It is also fashioned of plastic and is suspended by fine wire springs to a plastic speculum. The latter is in the shape of a truncated cone and possesses a scleral shoulder to provide additional support by the lids. In the case of both the Goldmann and the Allen-O'Brien lenses, only one reflecting surface is utilized and therefore only one quadrant of the angle may be visualized without rotating the lens about its antero-posterior axis.

In an effort to overcome this latter disadvantage, the Allen-Thorpe[14] gonioprism was devised. Here, a truncated pyramid is used, in which four totally reflecting prism surfaces are employed and the entire anterior chamber angle may be viewed with minimal rotation of the lens. It is also constructed of acrylic plastic, and is suspended in a plastic speculum to assist in proper positioning. A drop of one-percent methyl cellulose solution is placed on the smaller surface which contacts with the cornea. This surface has a radius of curvature of 8.2 mm. Since this is somewhat flatter than the cornea itself, slight pressure serves to bring the two surfaces into contact, eliminating the interference of air bubbles. The anterior surface is flat, and the reflecting surfaces are at an angle of 60 degrees to it. By proper adjustment, this lens can also be used for examination of the peripheral retina, as well as for observation of the vitreous and central retina through its central portion.

Many other modifications of the original indirect-vision lens of Goldmann have been proposed. Colenbrander[15] has suggested the use of a three-sided prism suspended in a wire support. Through this, one third of the anterior chamber angle may be viewed by direct observation, and the other two thirds, by means of totally reflecting prism surfaces, by indirect observation. A contact lens is also available for use with the Zeiss-Opton slitlamp which makes use of four mirrored surfaces for viewing the entire anterior chamber angle, and which is supported directly by the instrument mounting. A three-mirrored version of the Goldmann lens is now being manufactured, each reflecting surface being set at a different angle to the front plano surface, so that the anterior-chamber angle, peripheral retina, and posterior retina may be examined by rotating the lens.

The advantages of the indirect-vision lenses are numerous. More economical use of space and equipment is possible. There is no astigmatic distortion of the image because the emergent rays are normal to the plano front surface. By the same token, however, no inherent magnification is possessed by the lens itself. Because these lenses are used with the corneal microscope, rapid changes of magnification are possible, and certain portions of the angle can be more easily seen than with the direct-vision lens.

The indirect-vision lenses also have many points in their disfavor. Since they are made of plastic, they possess the same predisposition to scratching and the same poor wetting qualities which are mentioned above in connection with the plastic direct-vision lenses.

Since they are used with the patient in the erect position, the examiner must support the lens with one hand, and gonioscopy becomes difficult unless the biomicroscope being used possesses a coupled illuminating and magnifying system. Use of these lenses in the examination and treatment of cases of infantile glaucoma is not feasible, since the patient is in the supine position. Here, the Koeppe lens is ideal, for the findings made with it can be directly applied to the goniotomy lens of Barkan, should surgery be decided upon.

Binocular vision is difficult to maintain in the lateral directions when using the indirect-vision lens. This is because the two visual axes of the ordinary biomicroscope are on a horizontal plane while the longer axes of the rectangular reflecting surfaces used to view these quadrants are in the vertical direction. The superior and inferior quadrants are easier to view because the longer axis of the reflecting surface corresponds with the plane of the objectives. Further, unless the illuminating system of the slit-lamp is equipped with a device to rotate the slit beam, its advantages can be utilized in only a limited way, and diffuse illumination must be depended upon for much of the examination in the horizontal meridian.

D. Contact lens fluid

The use of the contact lens, of whatever type, necessitates the maintenance of optical continuity between the cornea and the lens. In other words, air must not be present between the two surfaces. For this reason fluid of some sort must be used between the two. For relatively short periods of observation, water is satisfactory, but if the lens is kept in place for some time, corneal edema with loss of transparency, as well as irritation, may supervene. Normal saline is much to be preferred because it is nonirritating and easily available. Some amount of care is required to avoid overfilling the contact lens, thus getting the saline on its outer surface. Should this occur a fine film of salt is left behind as evaporation proceeds, which interferes with good visibility. Only 0.06 cc. of solution is needed to fill the 16-mm. Koeppe lens.

A less irritating solution, if the eye is to be observed for longer periods of time, is the buffer solution of Gifford.[16] In 1945, Swan[17] proposed the use of a one-percent Methocel solution, which has about the same index of refraction as the cornea and is more viscous than the other solutions described. This latter fact makes its use advantageous where the patient is in the erect position, since there is less tendency for it to drain from behind the lens. The Methocel solution possesses the disadvantage of being difficult to remove from the lens should it be allowed to dry.

Various other agents have been advocated for use as contact lens fluid. Glycerine may be of value in some cases of edematous corneas, but it is much too irritating for routine use. Oils of various sorts have been used but are unsatisfactory for obvious reasons.

III. Magnifying devices

A. Ophthalmoscope

Salzmann,[2] in his early attempts to study the anterior chamber angle, used an ophthalmoscope as his magnifying device. Even as late as 1921, Troncoso[18] reported on the use of the electric ophthalmoscope in gonioscopy, the patient being in a supine position. Since the ophthalmoscope does not represent a telescopic system but is only a direct magnifier, its use is extremely limited and it is no longer employed.

B. The loupe

The principal difficulty with the use of any type of loupe is its relatively low magnifying ability. The simple Beebe loupe or the Bausch and Lomb Dualoupe produce a magnification of only two or three diameters. The newer telescopic loupes are little better from the standpoint of magnification although they are considerably less clumsy. The Zeiss-Gullstrand loupe possesses a magnification of ×2, and the Lempert-Storz, a variable mag-

nification from ×3 to ×6. With excellent lighting one can observe whether or not the angle is wide or narrow, and a large cyclodialysis cleft can be seen. Thorburn,[19] in 1927, reported on a study of 100 glaucoma cases using a ×4 loupe and a Koeppe lens. However, more recent refinements have made the use of the loupe inadequate for anything but the grossest type of gonioscopy.

C. Microscopes

The first adequate microscope for studying the anterior chamber angle was that devised, in 1925, by Troncoso.[4] This possessed a rotating prism at its tip to facilitate 360-degree observation, and had a magnification of either ×10 or ×20. It possessed a combined coaxial source of illumination, the power for which was provided by a battery in its handle. This monocular instrument was termed the "gonioscope," and is no longer manufactured.

Werner,[20] in 1932, first used the microscope removed from a slitlamp, which he supported in his hands, using a flashlight as his source of illumination. In order to give adequate support to this relatively heavy instrument so that sharp focus might be maintained, Barkan, Boyle, and Maisler,[21] in 1936, described a method of suspending a Zeiss microscope head from the ceiling by means of a telescoping tube with universal joints.

Since that time many varieties of supports have been proposed, which will be described subsequently. In 1941, Troncoso[22] described a new model of his original gonioscope. This is hand-held and possesses a pistol grip. It combines either a binocular or monocular microscope with a coaxial slit illuminator and has a magnification of either ×10 or ×20.

More recently a stripped-down version of the microscope used on the Haag-Streit (Goldmann) slitlamp has become available. The turret containing the objectives has been replaced by a slide containing only the ×1.6 objective and, when used with ×6 oculars, a total magnification of about ×10 is produced. Its total weight is approximately 14 ounces. If slightly higher magnification is desired, ×12 oculars can be substituted.

The magnification of most corneal microscopes ranges from ×10 to ×40 depending upon the objectives and oculars employed. A magnification of about ×20 is adequate for all but the most precise gonioscopy. With this degree of magnification, however, the depth of focus is extremely small and the slightest motion is sufficient to blur the image. For this reason the weight of the average slitlamp microscope renders it impractical as a hand-held instrument. The light weight of the hand-held Haag-Streit microscope makes it the most suitable, and, when used with a good light source and a glass Koeppe lens, it is adequate for all but the most exacting gonioscopy. It is probably the easiest of all methods for the beginner to use. If total magnifications in excess of ×15 are desired, no hand-held instrument can be maintained sufficiently steady for satisfactory use, and a more rigid support becomes necessary.

D. Microscope supports

If the indirect-vision type of contact lens is used in conjunction with a standard slitlamp, rigid support of the microscope is provided by the instrument itself. Any desired magnification may be obtained within the limits of the instrument, the maximum obtainable being approximately ×40 and the minimum about ×10. As previously mentioned, since one hand must be available for the contact lens, it is almost essential that an instrument with coupled illumination and magnifying systems be employed, rather than one of the older types.

Many varieties of supports have been described for use with heavier microscopes and when high magnifications are desired for use with the direct-vision lens and the patient in a supine position. The ceiling support of Barkan, Boyle, and Maisler[21] has already been mentioned. In 1938, Hartshorne[23] described the use of a Zeiss microscope head mounted on a camera tripod provided with

an extension bar. A somewhat similar mounting was described by Bogart,[24] in 1941. Alvaro and Silva,[25] in 1942, proposed the use of the adjustable base from a Zeiss slitlamp mounted on a small four-wheeled table. A universal joint was placed between the microscope head and the base to provide the necessary angulation.

Currently two excellent varieties of microscope supports are available for use in gonioscopy. The Barkan stand* is a tubular arrangement mounted on three wheels which can easily be moved around the patient. Side-to-side and to-and-fro movements are easily performed through the aid of universal joints. Up-and-down movements are controlled by a spring device which counterbalances the weight of the microscope. Considerable experience is required to control its motion easily and its use may present somewhat of a problem to the beginner. It does possess the advantage of mobility and, requiring only three square feet of floor space, can easily be stored in a corner of the examining room when not in use.

If floor space is at a premium, a tubular arrangement is obtainable by which the microscope can be suspended from the ceiling. This apparatus consists of two hollow aluminum tubes,† one sliding within the other, fastened by a universal joint to the ceiling directly over the head of the patient. A counterweight is enclosed within the inner tube and the microscope is attached to the lower tube by means of another universal joint. The up-and-down motility of this arrangement is much smoother than that afforded by the Barkan stand. A disadvantage present in early models is that the cable supporting the counterweight might become twisted and eventually break, leading to obvious difficulties. Care must also be taken that the tube and microscope, when swung out of use, are securely fastened to the wall, else the microscope may inadvertently swing in an arc directly toward the head of the patient being examined.

* The Barkan Stand is manufactured by the Parsons' Optical Laboratories, San Francisco, California.

† Manufactured by Parsons' Optical Laboratories and Jenkel-Davidson Optical Company of San Francisco, California.

IV. Illuminating systems

An adequate source of illumination is essential for good gonioscopy. The ophthalmoscope, flashlight, and reflecting mirrors which were utilized by the early investigators in this field were manifestly inadequate. The original gonioscope of Troncoso[4] was a step forward in the direction of providing a proper light source, but had the disadvantage of being battery powered and of low intensity. The use of the slitlamp illuminating device as described by Barkan, Boyle, and Maisler[21] provided excellent intensity of illumination, but possessed the disadvantage of inflexibility. The advantages of sharply focused illumination were ignored by others in favor of a fixed source of diffuse illumination such as could be provided by a standard operating light.

In 1936, Castroviejo[26] described an illuminating device which consisted of a lamp in a flexible vertical housing which could be mounted on the front of a detached slitlamp microscope. This possessed a totally reflecting prism which was located between the two objectives and provided a coaxial source of illumination. A modification of this was proposed by Fine,[27] in 1937. Here, the housing was horizontal rather than vertical and was designed to project through the central opening in the turret of the Zeiss binocular microscope. Both of these devices possessed the disadvantage of any coaxial illuminating system; namely, that the beam could not be deflected with respect to the axis of the microscope during the examination and further that a slit-type beam was not produced.

In 1941, Barkan[28] described a small, lightweight illuminating device which could be either mounted on the microscope or hand held to provide for flexibility of illumination. If desired, it could be mounted on a stand

of some type. It weighs five ounces and produces 500 foot-candles of illumination at five inches. It can also be focused to form a slit type of image of its filament. This illuminator has the advantages of brilliance, flexibility, and the ability to produce a slit-beam.

When using the indirect-vision contact lens with the patient seated at the slitlamp, the illuminating system of the slitlamp is utilized. Brilliant illumination is obtained with almost any model, but flexibility is considerably less than with the hand-held illuminator. In the majority of slitlamps, there is no provision for rotating the slit from vertical to horizontal so that diffuse illumination must be used in observing the medial and lateral angles. An attachment is available for use with the Zeiss-Opton slitlamp which accomplishes this rotation by means of a prism.

V. Summary and Conclusions

We have attempted to present a comprehensive survey of the various methods of gonioscopy, including the gradual development of the methods now in use from their beginnings some 50 years ago. A discussion of the three essential portions of the gonioscopic optical system has been presented together with some of the principles involved in each. The theoretical and practical advantages of the techniques to date have been mentioned.

On the basis of these observations, we have come to the conclusions that:

1. The easiest and least confusing gonioscopic method for beginners, and perhaps for the average ophthalmologist, is the use of a 16-mm. glass Koeppe contact lens, the Barkan hand illuminator, and the hand-held Haag-Streit gonioscopic microscope.

2. If space or finances for extra office equipment is at a premium, the Allen-Thorpe gonioprism, used with the slitlamp, is a satisfactory substitute. It has the added advantage of permitting fundus studies.

3. If more critical examination of the anterior chamber angle is desired, the use of high magnification with the glass Koeppe lens and the Barkan stand or telescopic microscope support becomes a necessity. This is particularly true for those with a special interest in the study of glaucoma, or those interested in doing goniotomy for infantile glaucoma.

4. The Goldmann contact lens is to be preferred for taking pictures of the angle of the anterior chamber.

In any case, it is of paramount importance that every ophthalmologist have one of the methods ready for use in his office. He should have gained sufficient experience to use this equipment easily and accurately, and to evaluate the information gained by it. If he fails to do so, the inevitable result will be faulty diagnosis, improper management, and some eyes unnecessarily lost.

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MODERN INDIRECT OPHTHALMOSCOPY*

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During recent years, there has been increasing interest in indirect ophthalmoscopy, mainly as the result of the development of a new type binocular self-luminous instrument.[1-3] This ophthalmoscope was devised primarily as a tool for the study and treatment of retinal detachment. The intense illumination system is of great value in those cases with hazy media. The indirect image, with less magnification, allows less distortion and a much larger field of view than that obtained with direct ophthalmoscopy. In searching for retinal breaks, especially in highly myopic eyes, this feature is a distinct advantage. When indirect ophthalmoscopy is done with the new type instrument mounted on the examiner's head, scleral depression can be easily performed. Originally described by Trantas,[4] and more recently by Schepens[5,6] scleral depression permits examination of the extreme fundus periphery including the ora serrata and posterior portions of the pars plana.

In spite of the many advantages offered by this method, there are certain difficulties which arise when indirect ophthalmoscopy is first attempted. These difficulties often discourage the examiner from attaining the skill necessary to perform the examination. It should be recalled that one's first attempt at direct ophthalmoscopy also left much to be desired. It is the purpose of this paper to describe the technique of indirect ophthalmoscopy as it is now used at the Retina Service of the Massachusetts Eye and Ear Infirmary, with special reference to the technique of scleral depression.

* From the Retina Foundation, Department of Ophthalmology of the Massachusetts Eye and Ear Infirmary and of Harvard Medical School. Paper 31, Retina Foundation.

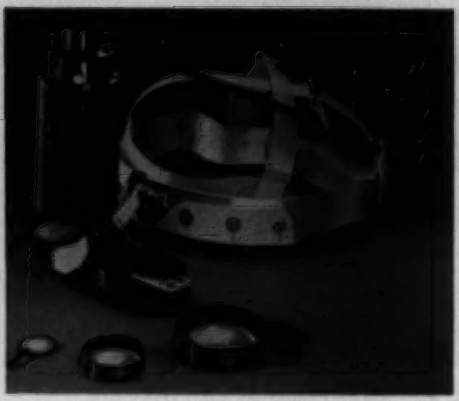

Fig. 1 (Brockhurst). The binocular indirect ophthalmoscope with three condensing lenses.

The equipment

A binocular indirect ophthalmoscope is now available,[7] and a brochure is supplied which describes the optical principles and use of the instrument.[8] Figure 1 shows the instrument as it is supplied with three condensing lenses. Ametropic examiners can have their own distance correction incorporated into the instrument, the correcting lenses being inserted under the eyepieces.

The condensing lens generally used is the +20D. plano-convex lens with a diameter of 35 mm. The working distance of this lens is about two inches from the patient's eye, which permits resting the hand on the patient's forehead or face so that the lens may be held steadily. The convex side of the lens should be held toward the observer in order to minimize reflections from the lens surfaces and distortion at the edges of the image. In contrast to direct ophthalmoscopy with a field of view of 10 to 17 degrees and magnification of about ×14, indirect ophthalmoscopy with the +20D. lens gives a field of view of about 37 degrees, the magnification being about ×3.

The +14D. lens with a diameter of 50 mm. results in more brilliant illumination of the fundus. Its working distance is about three inches from the patient's eye. The field of view with the +14D. lens is about 38 degrees, and the magnification is about ×4. The smaller +20D. lens with a diameter of 20 mm. has a small handle attached, and gives a field of view of about 22 degrees. This lens is convenient for use during surgery.

Right-handed examiners should hold the condensing lens in the left hand; the right hand is then free for drawing a picture of the fundus findings or for the use of a scleral depressor.

Scleral depression may be performed with a blunt instrument such as a muscle hook. A special instrument for scleral depression is now available,[9] which is attached to a thimble. By wearing the thimble on the middle finger, the examiner may use his index finger to aid in holding the eyelids.

The pupil

Good mydriasis is essential for good indirect ophthalmoscopy. A small pupil admits less light, and also makes positioning of the condensing lens more difficult. A parasympatholytic drug, for example two-percent homatropine, should be used in conjunction with a sympathomimetic drug, for example 10-percent neosynephrine. One drop of each repeated in 10 minutes usually results in wide dilatation after 30 minutes. Weaker mydriatics such as euphthalmine or paredrine are not effective as the intense illumination system will cause miosis. Cocaine should not be used for mydriatic purposes as corneal edema and hazing of the image results. Ointments similarly should not be used as the film of ointment on the cornea makes fundus examination indistinct.

In some cases, it may be necessary to give a subconjunctival injection in order to dilate the pupil maximally. Schepens[3] recommends use of the following mixture:

Neosynephrine hydrochloride	25 mg.
Homatropine bromhydrate	20 mg.
Procaine hydrochloride	50 mg.
Aqua Dist.	5 cc.

About 0.2 cc. is injected in each eye, mydriasis occurring in about 20 minutes.

Anxiety, circulatory disturbances, and vomiting which sometimes follow the subconjunctival injection of epinephrine have not been observed. In elderly patients, the mixture is safe as the blood pressure rises slowly.

The general fundus examination

The patient should be made comfortable as the examination is time consuming. Most patients can tolerate examinations of one hour or more if necessary; excited patients may be more co-operative if previously given a mild sedative. The examination should be done in a darkened room.

For examination of the posterior portions of the fundus both examiner and patient may sit facing one another; the drawing paper is then placed on the patient's knees. For examination of the fundus periphery it is best to have the patient lying down, for example, on a hospital litter.

Before discussing the technique of fundus examination, it is necessary to consider the problem of the inverted image which is obtained when using indirect ophthalmoscopy. The +20D. condensing lens which is held about two inches from the patient's eyes forms a real, inverted image of the fundus. This real image is located about two inches from the condensing lens on the observer's side. To minimize the confusion which results from dealing with an inverted image, it is suggested that the examiner stand behind the supine patient, as in doing intraocular surgery. When standing behind the patient at the 12-o'clock position, the examiner sees an inverted image of the fundus. But since the examiner is behind the patient, he is, in essence, inverted as he regards the patient. In other words, the examiner is observing an upside-down image from an upside-down position. The net effect is that the image as seen in the condensing lens has exactly the same relationships as an image obtained with direct ophthalmoscopy done in the conventional manner, that is, facing the patient. For example, if the right eye is being studied, the macula will appear on the examiner's left, the nervehead on his right, as he views the image in the condensing lens.

Until the patient's eye is light adapted, the strong illumination will cause orbicularis spasm and discomfort. For the first minute or two it is advantageous to use low illumination and examine the eye without using the condensing lens. A bright red reflex is obtained, and opacities in the cornea, lens, and vitreous can be studied.

When light adaptation has occurred, the light is directed into the pupil, and the patient is asked to look toward the light. A bright red reflex is seen. The +20D. condensing lens, convex side toward the observer, is placed in the path of the light about one inch from the patient's cornea. The lens is tilted slightly to displace reflections from its surfaces toward the edge of the lens. The examiner must insure that his own head, as well as the patient's eye, has not moved as the condensing lens is placed in position. A clear view of the macula and nervehead should be seen. By bringing the condensing lens away from the patient, the image will fill the condensing lens, the optimal working distance being about two inches from the patient's eye. A reflex from the cornea will be noted, this being reduced in size by moving the condensing lens either slightly toward or away from the patient. When examining other areas of the fundus, the corneal reflex is not visible.

When beginning this technique, the examiner may get too close to the condensing lens, this giving a larger but somewhat blurred image of the fundus, especially if the examiner is presbyopic. The observer's eyes should be about 14 to 16 inches from the condensing lens. A +2.0D. sphere is incorporated into the ophthalmoscope to assist the examiner in viewing the real image formed on his side of the condensing lens. If the image is blurred, the examiner should move his head still farther away from the condensing lens. Presbyopic examiners may require additional plus correction.

If diplopia occurs it usually can be readily corrected. Vertical diplopia is generally due to tilting of the ophthalmoscope. Horizontal diplopia results if the observer is too close to the patient. Examiners with high phorias at near can have suitable prisms incorporated in the instrument.

In order to see other areas of the fundus, it is necessary for the examiner to alter his own position, or the patient must change his position of gaze. Movement of the condensing lens alone will not permit examination of other fundus areas. It is helpful to have the patient fix his own finger with both eyes open if he has difficulty in holding his gaze steady. While the patient is fixing his finger, the finger may be moved by the examiner, fundus details being observed continuously. Although the examiner is behind the patient and sees an image with relationships similar to conventional direct ophthalmoscopy, he will be aware of the inverted character of the indirect image. As the patient looks upward toward the 12-o'clock position, the image, as seen in the condensing lens, moves downward toward the patient's feet. The patient is asked to look upward for examination of the upper fundus, downward for the lower fundus, temporally for the temporal areas, and nasally for study of the nasal areas.

It is helpful to make a drawing of the fundus details, showing the disc, macula, and major vessels which lead to retinal pathology.[3] The paper for the chart can be placed next to the supine patient's head, the 12-o'clock position of the chart directed toward the patient's feet, that is, inverted. The portion of the chart denoting the temporal half of the retina will then be nasal in relation to the patient's eye, and vice versa. Then the images as seen in the condensing lens are drawn on this chart. When completed and held with the 12-o'clock position toward the patient's head, the chart can be viewed as if it were a direct reproduction of the fundus, that is, similar to the image seen with direct ophthalmoscopy.

If the pupil dilates poorly, or if there is only a small opening in a pupillary membrane, a better image of the fundus will be obtained if the front edge of the ophthalmoscope mirror is tilted up about one to two mm. out of its normal position. This makes the illumination and observation systems more nearly parallel.

Examination of the fundus periphery

In order to examine the periphery in all quadrants, it is necessary to move around the patient's eye. For example, to examine the periphery at the 6-o'clock position, the observer must be at the 12-o'clock position, the patient gazing downward toward his own feet. In order to see the periphery at the 3-o'clock position, the examiner is at the 9-o'clock position, the patient gazing toward the 3-o'clock position. In order to make the examination less tiring for the observer, a high stool on casters may be used, allowing the examiner to sit and easily move around the patient's eye.

When the eye is rotated away from the examiner, so that the periphery may be seen, the shape of the pupil, as seen by the observer, is not round but an horizontal oval. This oval slit reduces the amount of light which can enter the eye, and also necessitates very accurate positioning of the condensing lens. To facilitate this portion of the examination, the illumination should be used at high intensity, and the front edge of the mirror should be tipped up slightly, as mentioned above.

The equatorial region can be seen easily and often a portion of the fundus anterior to the equator. Rarely, the ora serrata may be seen without scleral depression; in aphakia the ora can often be seen without scleral depression, especially in the zone of a full iridectomy. In order to see the region between equator and ora serrata, and portions of the pars plana, it is generally necessary to indent or depress the sclera over these areas. It is advisable to chart the major vessels to the equator before beginning this

portion of the examination. Then pathologic changes located in the extreme fundus periphery can be drawn in relation to these vessels.

The technique of scleral depression was originally suggested by Trantas,[4] who used direct ophthalmoscopy; he pressed on the globe with his thumbnail. The thimble depressor of Schepens[3,5] permits more accurate depression. It is usually worn on the middle finger of the right hand. The tip of the depressor is placed on the skin of the eyelid over the area of sclera to be indented. To facilitate application of the tip when examining the upper fundus periphery, the eyelid should be closed, the depressor tip being applied to the lid at the upper edge of the tarsus. When the patient opens the lid and looks up, the tip of the depressor can be easily slid under the orbital margin. The area to be indented is from six to 14 mm. behind the limbus. In eyes with normal intraocular pressure scleral depression is not painful, although it is helpful to tell the patient that he will feel slight pressure. In most retinal detachment cases, the intraocular pressure is below normal, and therefore scleral depression is easier to perform. The amount of pressure used to see the fundus periphery is very slight and of the same magnitude as one uses when estimating the intraocular pressure by palpation. A common error is to press too hard, especially if the examiner has difficulty in visualizing the fundus periphery.

Fig. 3 (Brockhurst). Correct method of scleral depression with tip applied tangential to curve of globe.

Many examiners make the mistake of pushing the depressor perpendicular against the globe in order to bring the periphery into view (fig. 2). This invariably causes pain and the patient squeezes his lids. It is better to use the scleral depressor tangential to the curve of the globe, flexing the finger slightly to cause indentation (fig. 3). The scleral depressor should be introduced as meridionally as possible. If introduced from the side (fig. 4)the tip, hidden by the lid, may not be in the meridian which is being observed, and the periphery will not be seen. When inserted in a direction closely parallel

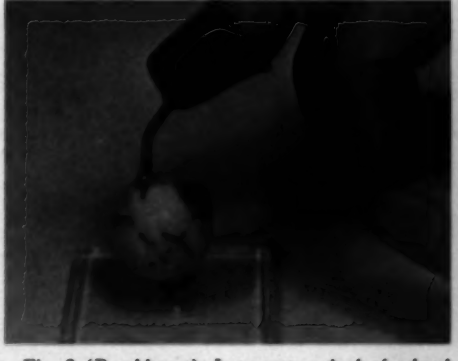

Fig. 2 (Brockhurst). Incorrect method of scleral depression with tip applied perpendicular to the globe.

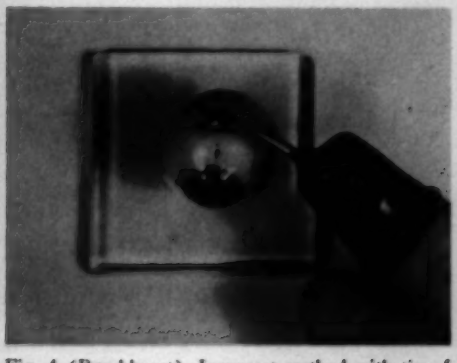

Fig. 4 (Brockhurst). Incorrect method with tip of scleral depressor introduced from side.

Fig. 5 (Brockhurst). Correct method with tip introduced close to the meridian of observation.

to the line of observation, the tip is more likely to be in the proper meridian (fig. 5).

The inferior fundus periphery is more difficult to see since the lower lid is generally tighter and many patients have difficulty in looking downward. The depressor tip should be placed on the skin of the lower lid about three to four mm. from the lid margin.

In order to depress the sclera at the 3- and 9-o'clock positions, in the palpebral fissure, it is usually necessary to apply pressure directly on the bulbar conjunctiva. Some patients tolerate this procedure well without local anesthesia, but in others it is best to anesthetize the conjunctiva with 0.5-percent tetracaine drops. If local anesthesia is necessary, it is best to do this portion of the examination last, as the tetracaine may cause edema of the corneal epithelium and hazing of the image.

When scleral depression is performed, the pressure tends to push the globe back toward the primary position of gaze. The patient is then urged to hold his eyes in extreme gaze more forcefully.

To obtain a good view of the extreme periphery four movable elements must be on a common axis and remain stationary. These are: (1) The examiner's eyes and ophthalmoscope, (2) the condensing lens, (3) the patient's eye, and (4) the scleral depressor tip (fig. 6). In general, there is no problem with the first two of these elements. Difficulties generally arise from improper positioning of the last two.

In regard to the patient, steady fixation of the eye is absolutely necessary. Some patients can hold their eyes in extreme positions of gaze without difficulty, but others have a great deal of trouble with this portion of the examination. For the latter group, it helps to have the patient observe his own finger. Even if the patient is unable actually to see his finger, he knows by proprioception where the finger is located. The ex-

Fig. 6 (Brockhurst). The ophthalmoscope, condensing lens, patient's eye, and depressor tip must lie on the same axis.

aminer can move the patient's finger to any location he wishes.

The fourth factor, that of the scleral depressor, is the most troublesome. Commonly, the tip of the depressor is not in the meridian which is being illuminated and observed. Introduction of the depressor close to the line of observation will help solve this problem. The other difficulty is the correct depth of the depressor tip, that is, the distance behind the limbus at which to indent. The most satisfactory way to approach this problem may be outlined as follows:

1. The patient is asked to turn his eyes away from the examiner maximally so that the pupil appears as an horizontal oval, almost slit-shaped. For example, the patient is asked to look up in the 12-o'clock meridian.

2. The examiner, stationed at the 6-o'clock position, directs the ophthalmoscope beam of light into the pupil.

3. The condensing lens is held in the left hand about two inches from the patient's eye, but outside of the beam of light, to the left.

4. The scleral depressor, introduced in the lid fold before the patient looked up, is adjusted so that the tip is held tangential to the curve of the globe and as close as possible to the meridian being illuminated. The tip of the depressor should be about four to five mm. behind the limbus. The finger holding the depressor is gently flexed, causing slight pressure on the sclera, until the ciliary processes are seen. The depressor tip is then moved posteriorly in the same meridian, slight pressure being maintained. The red reflex seen in the oval pupil will turn gray when the tip of the depressor is about seven mm. behind the limbus. Hold the depressor steady when the gray appearance is noted.

5. The condensing lens is inserted in the beam of light about two inches in front of the patient's cornea. The lens should be slightly tilted so that its plane approaches the plane of the pupil, that is, the upper edge of the lens is farther from the examiner. A clear view of the indented area should be seen in the condensing lens, the teeth of the ora serrata pointing toward the lower edge of the lens. The depressor tip may be moved slightly from side to side, and fore and aft, to bring neighboring areas into view. Some examiners prefer to approach the ora serrata region from its posterior aspect, placing the tip of the depressor about 10 to 11 mm. behind the limbus and sliding it forward until the red reflex turns gray. In order to examine the periphery in other meridians, it is necessary to reposition the patient's eye, and also to alter the examiner's position.

The complete examination of the fundus periphery with scleral depression is no more dangerous than are pupillary dilatation and tonometry. No accidents have been observed after repeated examinations of several thousand patients. The possibility that scleral depression may cause retinal breaks appears to be entirely unfounded.

Summary

The technique of indirect ophthalmoscopy is discussed with special reference to the use of scleral depression and examination of the fundus periphery.

327 Charles Street (14).

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A SAFE SECTION FOR CATARACT EXTRACTION*

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All are familiar with the difficulties and hazards of a full Graefe section. The difficulties and hazards increase if preplaced sutures, particularly gut sutures, and a limbal-based conjunctival flap are used.

To avoid these hazards, some surgeons make a small Graefe section and enlarge it with scissors. Other surgeons use a keratome to make the initial incision and enlarge it with scissors. With a keratome it is easier and safer for the average surgeon to make the preliminary incision in the proper place. However, introducing any sharp-pointed knife several millimeters into the anterior chamber is not without risk.

It seems unnecessary to review the numerous types of incision which have been advocated for cataract extraction or the many kinds of knives that have been devised for making the section.

A soft eye has proved to be highly desirable for intracapsular cataract extractions and is an important defense against vitreous loss and other complications. The pronounced hypotony, which is obtained by a retrobulbar injection of an anesthetic solution with epinephrine and hyaluronidase followed by firm pressure over the globe, alters the mechanics of the extraction, but with a little practice the extraction is easier and much safer.

To facilitate making a good, clean, nonpenetrating incision of the desired length and depth for preplaced sutures, when the eye is soft, a different type of instrument (fig. 1) which has been called a scleratome is used. It looks like a cystotome but it is heavier and has a blade 0.75 to 1.0 mm. in length. The length of the blade and shaft prevents making a perforating incision if the shaft is held firmly against the globe (fig. 2-A, insert). However, no harm is done if a perforating incision is made. The shaft of the instrument is bent at an angle of 45 degrees which facilitates making a nonperforating incision.

A conjunctival flap is prepared in the usual manner. If a limbal-based flap is used, the corneoscleral area where the incision is to be made should be bare of conjunctiva and Tenon's capsule; otherwise the conjunctival flap tends to be drawn in front of the blade as the incision is made.

To make a satisfactory incision of the proper length and depth in one stroke with this instrument, *firm double fixation is essential*. With firm double fixation, it is easy. Different types of double fixation forceps were tried, but the following method which was suggested by my associate, Dr. Ken-

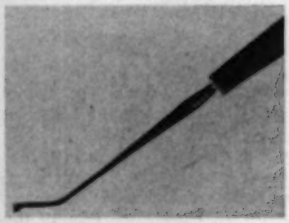

Fig. 1 (Atkinson). Knife for making incision, blade is 0.75 to 1.0 mm. long, shaft is bent at angle of 45 degrees.

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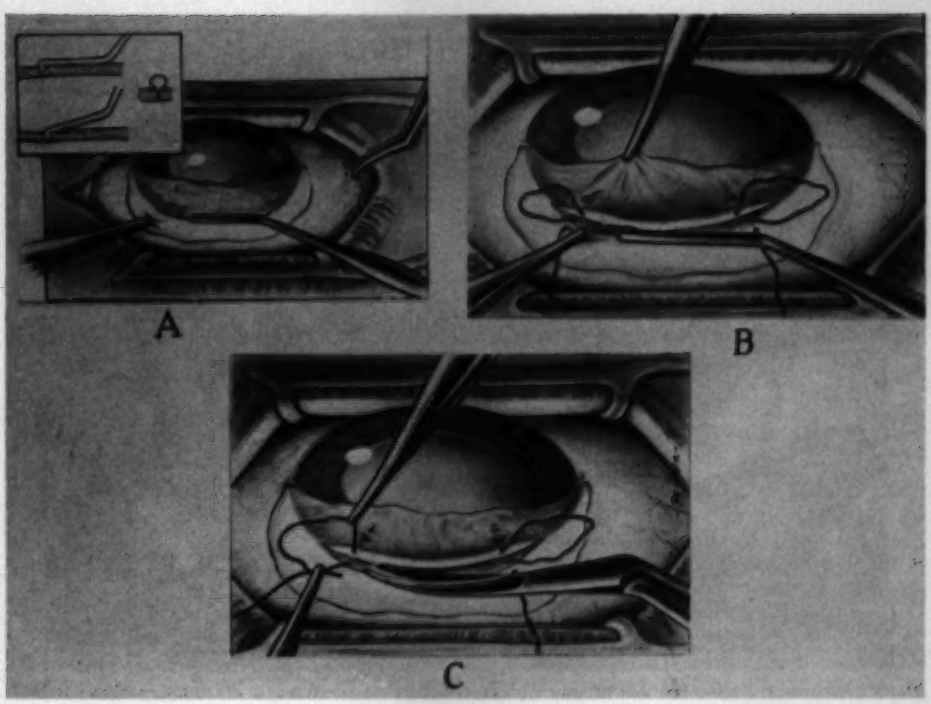

Fig. 2 (Atkinson). (A-insert) shows shaft held firmly against globe to prevent perforating and tipped up to make perforating incision. (A) Firm double fixation. (B) Upper lip held by surgeon and conjunctival flap by assistant to make perforating incision. (C) Suture held out of way by assistant while wound is enlarged with scissors.

nedy, seems to be the most satisfactory.

The surgeon uses sharp-toothed forceps to obtain a firm bite of episcleral tissue near where the incision is to begin (fig. 2-A). The assistant grasps the tendon of the rectus muscle at its insertion on the opposite side of the globe with fixation forceps. The blade of the knife is then introduced to its full length and at an angle which will produce the desired bevel. The shaft of the instrument is held *firmly* against the globe and the incision is made slowly. When the shaft of the instrument is held *firmly* against the globe and the blade is of the proper length,* a good clean incision of the desired length and depth can be made easily with one stroke. Making the incision on the left eye with the right hand, when standing at the head of the table, there is a tendency to elevate the shaft and produce a perforating incision particularly if the patient's nose is prominent. This can be overcome by rotating the globe temporally or bending the shaft of the instrument to a more acute angle than 45 degrees. If the instrument is tipped up so that the shaft is not in contact with the globe, then the blade will cut deeper (fig. 2-A, insert) and a perforating incision can be made. The point of the instrument barely enters the anterior chamber so that it does not come in contact with the iris and when the eye is soft, very little or no aqueous escapes.

It is as easy to introduce sutures after making a perforating incision in this manner as after a keratome incision. However, I prefer to introduce the sutures before an

* The length of the blade may vary somewhat and it is usually shorter after it has been resharpened. If a long, beveled incision is to be made, a longer blade may be required than if the incision is to be made at right angles to the corneal surface.

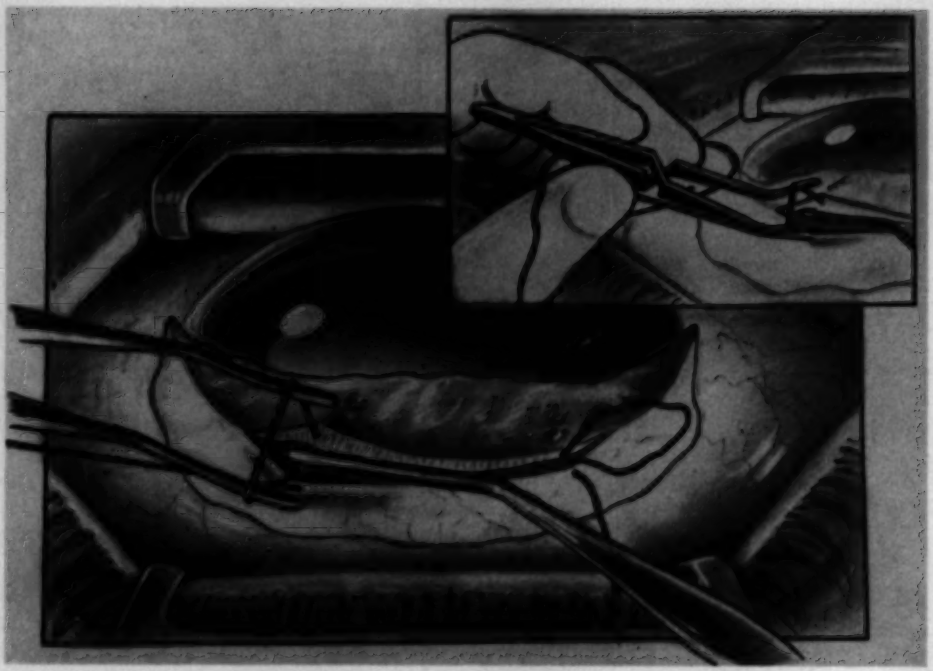

Fig. 3 (Atkinson). Suture held out of way with cross-action capsule forceps, which also provides fixation for making perforation incision if a good assistant is not available.

opening is made into the anterior chamber because it is easier for me to place them accurately.

After a long deep nonpenetrating incision is made, 6-0 mild chromic (Ethicon) gut sutures are introduced (fig. 2-B). On the end of each suture is a double figure-eight knot to prevent the suture from being pulled through the cornea. Having the knot pulled up against the cornea makes it easier to hold the loop out of the way when enlarging with scissors. With only one loop the suture can be pulled up to close the wound quickly and securely in case of emergency. The long end of the suture can then be held by the assistant; if necessary, to keep the wound tightly closed while the sutures are passed through the conjunctiva to be tied on the corneal or conjunctival surface following the extraction.

After the stutures are introduced, a perforating incision is made. To do this, the upper lip of the preliminary incision is grasped by the surgeon and the conjunctival flap is held by the assistant (fig. 2-B). In this way, both lips of the nonpenetrating incision are held apart and a tenting of the wound is produced. The same knife is then introduced at the bottom of the incision (fig. 2-B) and after the point enters the anterior chamber the shaft is raised, which tips the point away from the iris so that it barely projects into the anterior chamber. The penetrating incision can be made between the sutures long enough to introduce the scissors with which the wound is enlarged in the same manner as following a small Graefe or keratome incision. The sutures are held out of the way by the assistant (fig. 2-C) while the incision is enlarged at the site of the sutures and then continued to the 180-degree meridian. If a good assistant is not available, the surgeon can hold the loop of the suture out of the way with cross action capsule forceps while the perforating incision is made, and

when the wound is enlarged with scissors. The blades of the forceps are passed through the loop. The long arm of the suture, on the end of which the needle is attached, is held against the side of the forceps with thumb or finger to control the size of the loop. Pressure to open the forceps holds the arm of the suture. As the blades of the forceps open, they spread the loop, separate the edges of the wound, and provide firm fixation to make the incision with the scleratome at the site of the suture (fig. 3). The wound is then enlarged with scissors.

If the incision is made with a good bevel in avascular cornea, it is farther from the root of the iris, which makes it easier to enlarge with scissors without injuring the iris. There is less bleeding at the time of operation and during convalescence, and a leak or iris prolapse is less likely to occur.

One objection to this method of making the section, which has been observed after using it for over a year, is that it eliminates some of the thrill of the cataract extraction. However, this is compensated for by having the pleasure of seeing more good-looking aphakic eyes after the operation.

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THE ELECTROMYOGRAM OF THE LATERAL RECTUS MUSCLE*

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The purpose of this article is to report a method of obtaining the electromyogram of the lateral rectus of the human being.

I have recorded action potentials from my own lateral recti muscles two times. I will have little opportunity in the near future of repeating the experiment under such laboratory conditions as have been used and for this reason, I feel, the records should be made available now. By themselves, the records are not conclusive, but interpreted against a background of many experiments with the rabbit under similar favorable conditions, much useful information can be obtained.

Björk (1952),[1] Björk and Kugelberg (1953a, 1953b),[2,3] and Adler (1953)[4] reported on the electromyogram of the extraocular muscles in the human. Björk's method employs one monopolar needle electrode on the muscle in question and the other not on the muscle. Adler states, "Using insulated bipolar electrodes inserted into one of our lateral rectus muscles, we recorded the changes in electrical potential, and the number of impulses discharged per second in this muscle in varying positions of gaze."

My method places two monopolar electrodes in the muscle to be studied; this has advantages. There can be no doubt as to the origin of the potentials—a consideration of theoretic importance. Free movement of the globe is possible. In addition, the experiments were done on a trained observer with an entirely normal visual apparatus. (Note: Wire electrodes do not give records with discrete motor units showing, such as are seen with needle electrodes.)

METHOD

The eye was draped for operation and an aseptic technique followed. Using two-percent tetracaine local anesthesia, two wire electrodes were introduced into the lateral rectus muscle. The one nearest the cornea was placed near the beginning of the muscle belly (14 mm. behind the limbus) and the other about five mm. behind the first. The wire electrodes were 40-gauge enamel-covered copper wire denuded at a suitable point

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for about two mm. The lead nearest the cornea was the indifferent lead and the other, the active lead. For recording, a Dumont oscilloscope Model 322 with a preamplifier was used. From the tube face, the tracings were photographed with a Grass kymograph camera. For details of the method see Magee (1954).[5]

During the experiment the subject was seated with the head fixed in a mount.

The motor unit

Quantitative descriptions of motor units of eye muscles are not numerous. Lorente de Nó (1935),[6] while discussing another subject, shows records of the rabbit lateral rectus muscle during stimulation of the sixth nerve. Some idea can be obtained from these records of the duration of the discharge of the motor unit of the rabbit, approximately 8.0 msec. for the diphasic response. Pulfrich (1952)[7] and Reid (1949)[8] show motor unit responses but they are difficult to assess quantitatively. Björk (1952)[1] and Björk and Kugelberg (1953a, 1953b),[2,3] report the aplitude in the human to be 20 to 150 microvolts (μv.) and one to two msec. in duration.

In resting records from the horizontal recti in rabbits the smallest element that can be identified as an action potential is found to be about four msec. in duration (monophasic) and about 20 μv. in amplitude. Figure 1 shows this: it is the usual record obtained from the rabbit. (For details of the method see Magee [1954].[5])

Many variables are to be considered but the record is more or less the same whenever done. The position of the electrodes in the muscle, movements of the eyes, trauma on introduction of the electrodes, the size of the denuded area of the electrode and its relation to firing motor units, and other factors are important. The further apart the electrodes, the greater the amplitude is the gen-

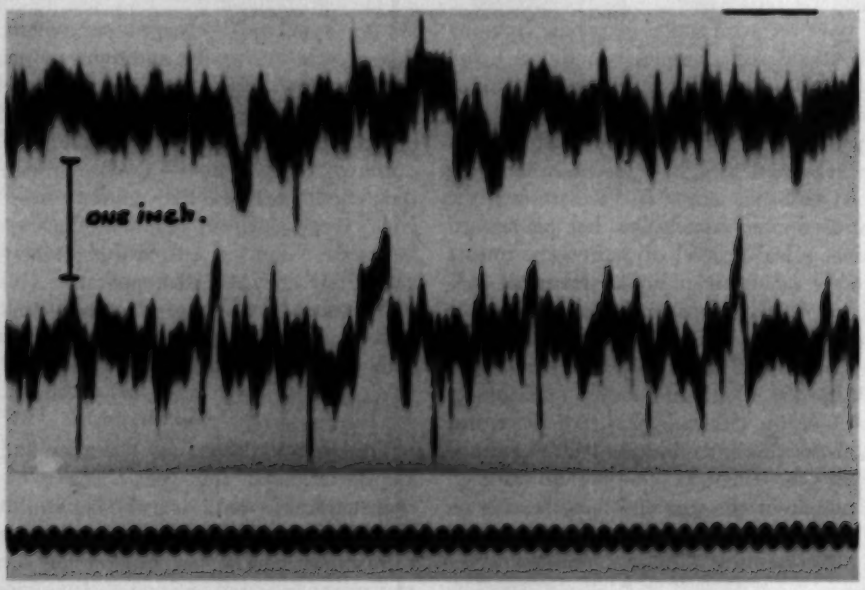


Fig. 1 (Magee).* The right eye of the rabbit with simultaneous recording from the right medial rectus, and right lateral rectus at rest. Upper channel right lateral rectus; lower, right medial rectus. Gain 16μv./in.; speed of film 100 mm./sec.; signal 100 cps.

* All records read from right to left.

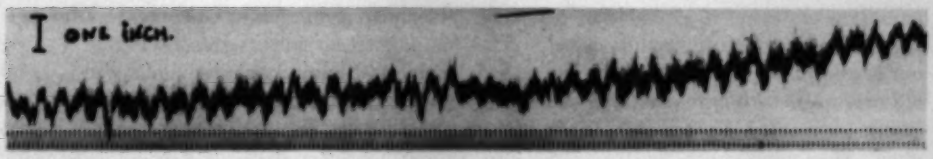


Fig. 2A (Magee). The right lateral rectus of the human during optokinetic nystagmus. Drum rotation counterclockwise. Film speed 250 mm./sec.; 30μv./in.; time signal 250 cps.

eral rule. While observing the tracing on the face of the tube, the record is seen to vary continuously.

Of the various types of activity recorded from the lateral rectus of the human, a good place to study the motor unit seems to be during optokinetic nystagmus. Unlike vestibular nystagmus of the rabbit, optokinetic nystagmus recorded here is not exactly periodic but irregular (fig. 2a). (Note: Sixty cycle interference appears in some of the records—it is important to ignore it in the interpretation.) It is obvious that there are many action potentials being recorded in the segment shown: likewise we might assume that the areas of inactivity interspersed between areas of motor unit firing are times of minimal motor unit firing or perhaps even inhibition. The element added to the quiet areas no doubt represents firing of motor units.

The quantitative aspects of the problem come next. (Note: Needle electrodes are more suitable for study of the motor units,

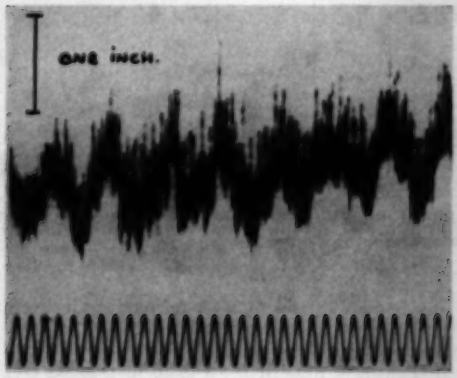


Fig. 2B (Magee). Enlarged portion of Figure 2A to show details of spikes.

but the wire electrodes possess certain advantages in recording.) Assuming that the discrete spikes are motor units firing, it would appear that the motor unit of the extraocular muscles of the human is from about 15 μv. to several times this in amplitude and about one msec. in duration. Considering the anatomy of these structures, this finding might not be inconsistent for supposedly the ratio of nerve fibers to muscle fibers is low in eye muscles (Tergast 1873,[9] Bors 1925-1926[10]). Neverthless, the figures given are small indeed—much smaller than those given for other types of muscle tissue (Clark 1930[11]) except perhaps smooth muscle.

Electromyogram of extraocular muscles at rest

The electromyogram of the rabbit eye at rest has been done many times by me including simultaneous recording of the horizontal recti of one eye. The record is about the same every time except for small variations due to the factors mentioned above. There is usually firing of several motor units; in addition, the isoelectric line is wavy. During simultaneous recording of antagonists (fig. 1) there appears to be a constant firing of motor units to a small extent. The two simultaneous tracings appear different; the difference is probably due to a greater amount of motor unit firing in the medial rectus. Certainly, there is no interval of quiet or inhibition followed by motor unit firing in one muscle with corresponding firing and inhibition in the other. Rather, there is some constant firing in each muscle when the eye is at rest.

Of my records of human beings, just the

lateral rectus alone is available at rest. The subject is gazing at infinity binocularly without fixating anything specifically. The record is fairly quiet with minimal motor unit firing in one segment and presumably none in another. Waviness, characteristic of the electromyogram of the extraocular muscles, is present (exclusive of 60-cycle interference). (Note: Using this particular method of recording.)

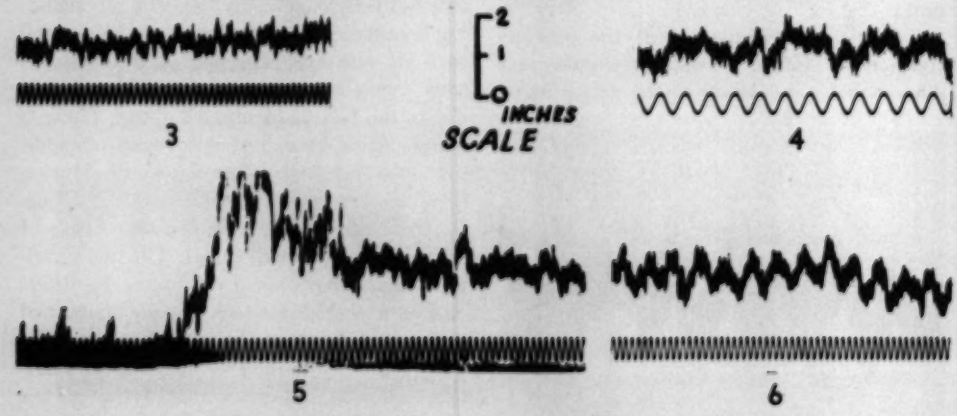
When considering the eye other than at rest, more than one type of muscle contraction exists. There is the activity of the extraocular muscles when the eye is fixing an object; after a fashion this might be called a type of isometric contraction. When the globe is moving from one position to another, one might say that the extraocular muscles are undergoing isotonic contraction (the contracting muscle at any rate). These are terms used in muscle physiology in general and how well they apply to the extraocular muscles is an open question.

During fixation

I will consider the eye during fixation first and, later, while moving grossly. Figure 3 shows the right eye fixating an object binocularly straight ahead (0 degrees) as far as it is possible to line up grossly. Figure 4 is the same as Figure 3 only the film speed is 1,000 mm./sec. There is motor unit firing. In contrast Figure 5 shows large spikes. The motor unit firing is greater in Figure 4 than in the record at rest (fig. 6). In Figure 3 the conditions are the same as for Figure 4 except that the film speed is one-fourth less. By comparing Figures 3 and 4 it can be seen that part of the waviness is probably due to variations in the isoelectric line, and this in turn is ultimately due most likely to movements of the extraocular muscles themselves. This waviness seems characteristic of this method of recording.

When the eye fixes, it might very well be that the muscles if not the eye itself undergo small movements, however small. The fact that motor units are firing and that there are variations in the isoelectric line constantly would point to this. In the rabbit, both these elements can be eliminated by retrobulbar injection of procaine, indicating they are a local phenomenon (Magee, 1954[8]).

Next, I will present records of the eye fixing in varying amounts of abduction and



Figs. 3-6 (Magee). (Fig. 3). Isometric contraction: fixing zero-degrees right lateral rectus; film speed 250 mm./sec.; signal 250 cps.; gain 30 μv./in. (Fig. 4). Isometric contraction: fixing zero-degrees right lateral rectus; film speed 1,000 mm./sec.; gain 30 μv./in.; signal 250 cps. (Fig. 5). Right lateral rectus of the human during convergence. The subject has just changed fixation from 2,850 mm. to 63 mm. marked by the black line superimposed on the time signal. Gain is 30 μv./in.; film speed 250 mm./sec. and the signal 250 cps. (Fig. 6A). Right lateral rectus of human while gazing at infinity. There are few spikes in this record. Time signal 250 cps.; gain 30 μv./in.; speed of film 250 mm./sec.

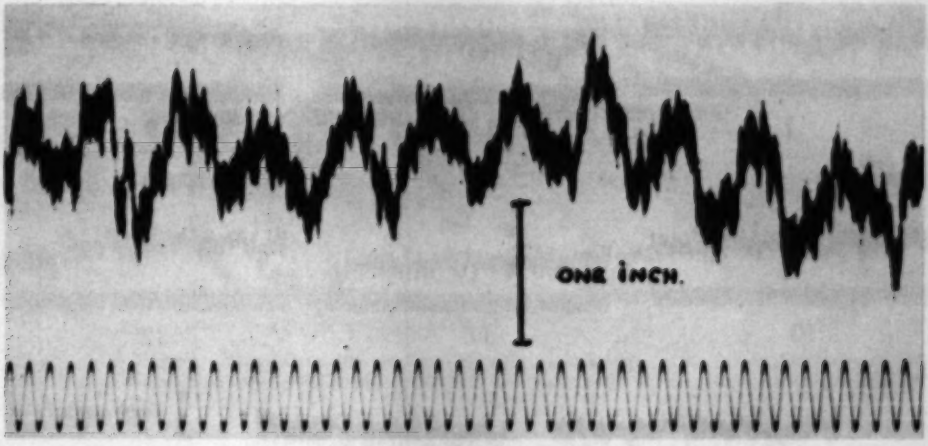


Fig. 6B (Magee). Enlarged portion of Figure 6A.

adduction—we might say the isometric type of contraction. Because the differences in the records in varying amounts of abduction and adduction are small they are difficult of demonstration especially with small segments of the tracing. Neverthless, the more motor unit activity is present, the greater the amount of abduction. Likewise, the isoelectric line is more wavy. Compare Figures 7, 8, 9, 10 and 3.

In isometric adduction of the right eye, the recording from the lateral rectus does not show much in the varying amounts of adduction. Figures 11, 12, 13, and 14 are not too dissimilar. Because of the irregularity of the isoelectric line, quantitative estimation is not easy. It would seem that motor unit firing is about the same in adduction of varying amounts. Irregularity of the isoelectric line is about the same in the different grades of adduction shown.

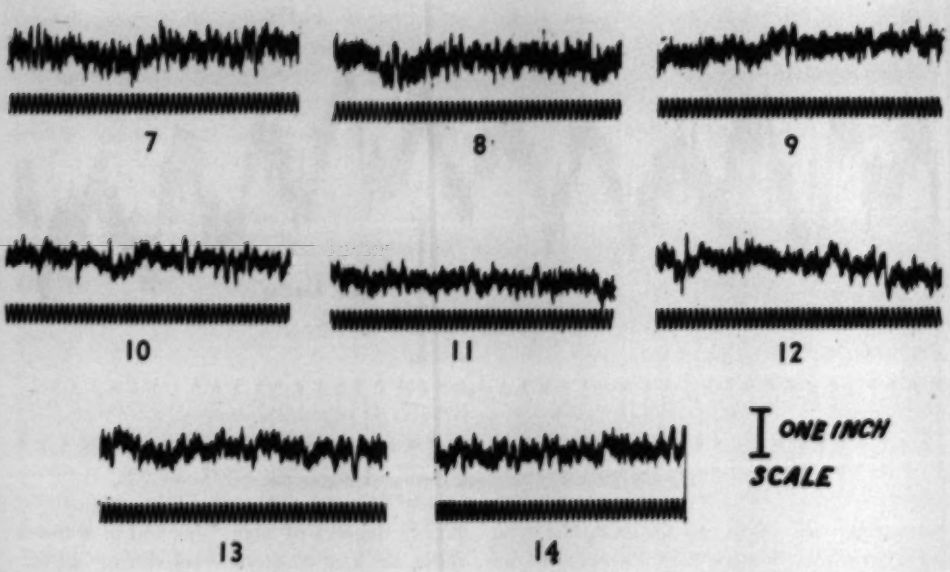
In comparing isometric abduction with adduction, the following should be pointed out. First of all, the irregularity of the isoelectric line does not vary greatly in all phases of both. The difference is small, and, if anything, the irregularity of the isoelectric line is greatest during large amounts of abduction and least during large amounts of adduction. Compare Figures 7 and 11. Likewise, motor unit firing is greatest during the higher degrees of abduction and at a lower more or less constant level during adduction. It is difficult to assess the amount of motor unit firing during adduction; it appears about the same in all amounts.

Continuing on with the extraocular muscles other than at rest, in contrast to the eye when fixating or during isometric contraction, there is the response of the muscle when the eye is moving grossly. We might call this an isotonic type of contraction.

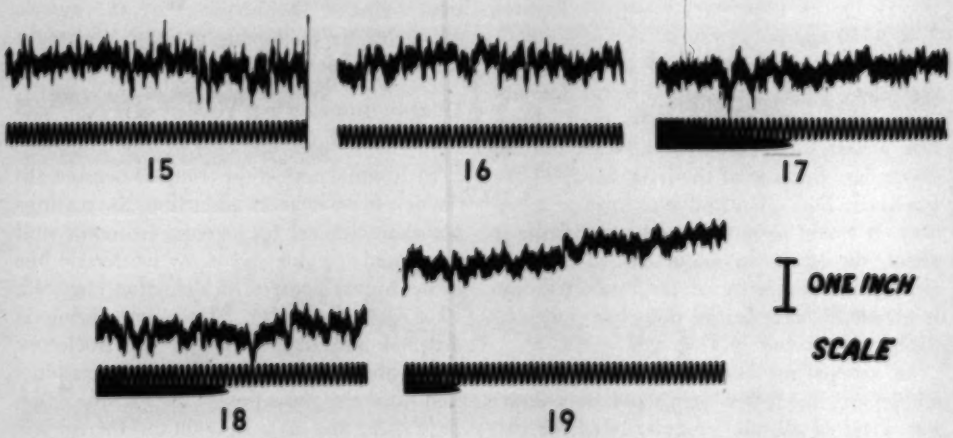
During horizontal voluntary movement of the globe

In isotonic adduction from 40 degrees abduction to 40 degrees adduction, the tracings are characterized by increase in motor unit firing and irregularity of the isoelectric line in the higher degrees of abduction (figs. 15, 16, 17, 18, and 19). Motor unit firing is minimal and irregularity of the isoelectric line is about the same during isotonic adduction once the globe passes the midline (figs. 20, 21, 22, and 23). In addition, the isoelectric line shows marked gross variations related probably to large movements of the muscle. These gross movements are most in about 20 degrees' adduction.

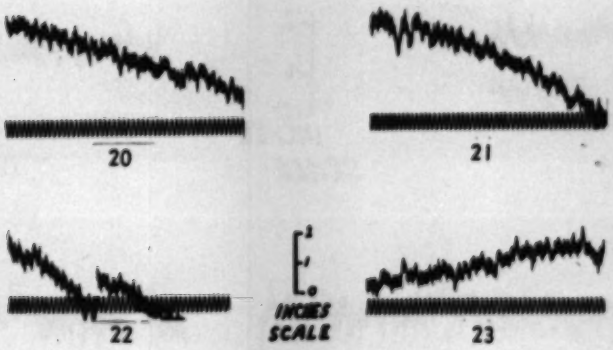
Isotonic abduction, the reverse of the above, is similar, showing the greatest motor unit firing and irregularity of the isoelectric



Figs. 7-14 (Magee). Figs. 7, 8, 9, and 10 show the response of the right lateral rectus during 40, 30, 20, and 10 degrees isometric abduction. Film speed 250 mm./sec.; signal 250 cps.; gain 30 μv./in. Note how the motor unit firing in particular decreases as abduction decreases. The waviness of the isoelectric line decreases somewhat also. Figs. 11, 12, 13, and 14 show the response of the right lateral rectus during 40, 30, 20, and 10 degrees isometric adduction, respectively. Film speed, gain, and signal as in Figure 10. Motor unit firing is not great and is about the same in the four records. Likewise, waviness in the isoelectric line is about the same in the various degrees of adduction.



Figs. 15-19 (Magee). These pictures show the response from the right lateral rectus during movement of the globe (isotonic adduction) from 40-degrees abduction to zero degrees in 10-degree stages, that is, 40, 30, 20, 10, and zero-degrees, respectively. Film speed, signal, and gain as in Figure 10. Motor unit firing decreases as abduction decreases; waviness is greater apparently in the greater amounts of abduction.



Figs. 20-23 (Magee). These show the response from the right lateral rectus during movement of the globe (isotonic adduction) from 10, 20, 30, and 40 degrees, respectively. Film speed, gain, and signal as in Figure 10.

line in the greater degrees of abduction. From the position of 40 degrees' adduction to the midline, or zero degree position, the recordings are about the same. As in the case of isotonic adduction, the gross movements of the isoelectric line also occur here at the position of about 10 degrees' adduction.

During elevation and depression of the globe

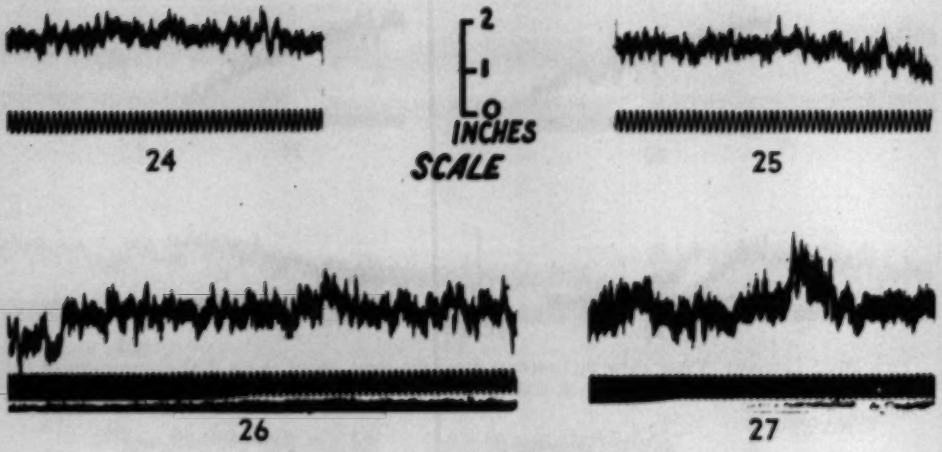
Still another type of activity of the extraocular muscles, other than at rest, is the record of the right lateral rectus in eyes up and down (one meter up or down at a distance of 2.85 meters) during isometric contraction. In the eyes-down position, there is minimal motor unit firing and less irregularity of the isoelectric line than in fixing straight ahead. See Figure 24 and compare with Figure 3. It seems from Figure 24 that the lateral rectus plays a minimal part in maintaining depression of the globe.

In the eyes-up position, during isometric contraction, the right lateral rectus record shows more irregularity of the isoelectric line than in the eyes-down position (fig. 25). The motor unit firing appears, if anything, more in eyes up than in eyes down.

Isotonic contraction from eyes straight to eyes down and eyes up is shown in Figures 26 and 27 respectively. The black mark on the bottom of the record indicates the time the subject began to look down or up from eyes straight. Considering the subject to have a latent period of a 10th of a second, the actual beginning of the activity from eyes straight is 100 msec. earlier than shown. It appears that motor unit firing in the right lateral rectus is greater in eyes up than down during isotonic contraction. Furthermore, there is more motor unit firing in the right lateral rectus during depression or elevation of the globe than when once it has been depressed as described. Waviness of the isoelectric line appears least during isotonic contraction in eyes up.

During convergence

Convergence and divergence are still other types of extraocular muscle activity other than at rest. Using a light to mark the film as described above to indicate the beginning of convergence and divergence the results are shown (the distance was from 2,850 to 63 mm. and vice versa). Figures 5 and 28 show the onset of convergence and after the eyes have stabilized somewhat in the convergent position respectively. It appears that the lateral rectus contracts somewhat early in convergence presumably against strong contraction of the medial rectus. Likewise, the position of convergence seems to be reached only after some sort of oscillating process as indicated by Figure 28. This so-called oscillating process can be seen grossly on convergence. I have only one record of



Figs. 24-27 Magee). (Fig. 24). Human subject fixing an object 1,000 mm. below the horizontal at 2,850 mm. Gain, signal, film speed as in Figure 10. (Fig. 25). Human subject fixating 1,000 mm. above the horizontal at 2,850 mm. distance. Gain 30 μv./in.; signal 250 cps.; film speed at 250 mm./sec. (Figs. 26 and 27). Human subject, right lateral rectus during fixation at distances as in Figure 25 (isotonic contraction not isometric) from eyes straight to eyes down (fig. 26) and from eyes straight to eyes up (fig. 27). Gain, film speed, and signal as in Figure 25.

convergence as described; one other that I have shows only marked variation of the isoelectric line.

During divergence

There are four records of divergence done as described for convergence, only in reversed fixation. In all four records, the onset is characterized only by marked variation of the isoelectric line with no outstanding motor unit activity (fig. 29). In three of the four records, there is marked segmental motor unit activity presumably when the divergent position is nearing stabilization (fig. 30).

During optokinetic nystagmus

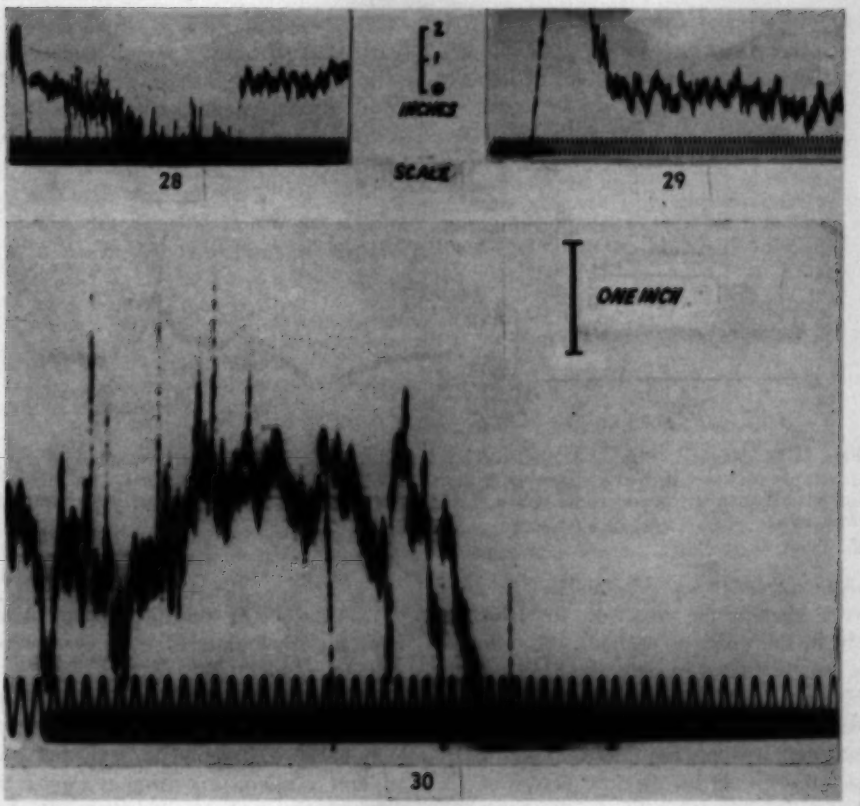
Optokinetic nystagmus has been mentioned before. A drum six inches in diameter and six inches high with alternating black and white stripes one-inch wide was used. Records were made rotating the drum (about 25 cm. in front of the subject at about 40 r.p.m.) either in a clockwise or counterclockwise fashion. This gives the quick and slow phase with the right eye respectively.

Records of the slow phase are satisfactory (for reasons difficult to explain now 60-cycle interference is apparent) and are characteristic. There is marked firing of motor units with decreased irregularity of the isoelectric line. Such a segment is interspersed with shorter segments showing little or no motor unit activity and some irregularity of the isoelectric line (fig. 2). The length of the slow phase varies considerably as does the representation of the quick phase in counterclockwise rotation of the drum while recording from the right lateral rectus in the human.

The records during clockwise rotation of the drum cannot be verified so they will not be presented.

During head rotation

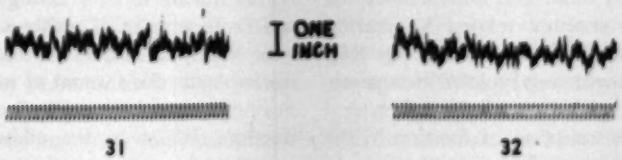
The last type of activity to be described is tracings made from the right lateral rectus with the eyes fixing the zero-degrees position while rotating the head first right and then left. (Note: This method of study was recommended by R. N. Berke, M.D.) Figure 31 shows the record during rotation of the head to the right, and Figure 32 dur-



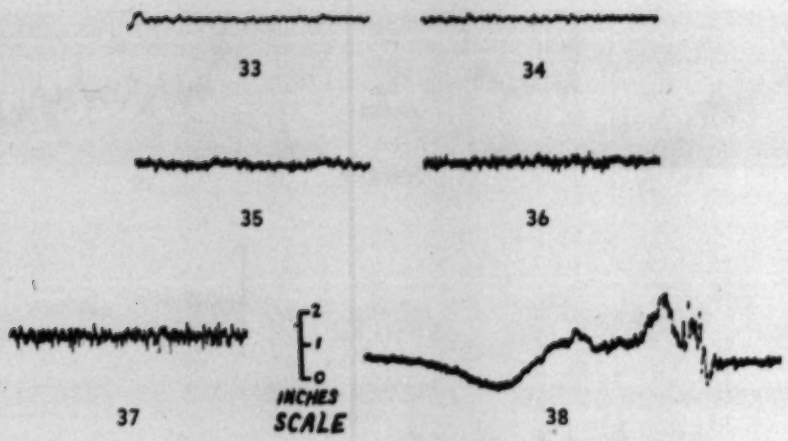
Figs. 28-30 (Magee). (Fig. 28). Human subject right lateral rectus; fixation from 2,850 to 63 mm., showing oscillating muscle response toward the end of convergence. Gain, film speed, and signal as in Figure 25. (Fig. 29). Human subject right lateral rectus fixating from 63 mm. to 2,850 mm. on marking with light. Gain, film speed, signal as in Figure 25. (Fig. 30). Toward termination of divergence, as stabilization is being reached.

ing rotation of the head to the left. These records are unsatisfactory for the most part but one is shown to indicate that the technique has possibilities. In fact, if clinical application is to be made, this would be worth further study.

Figures 33, 34, 35, 36, and 37 show the recordings made from my left lateral rectus on an occasion previous to the experiment described in detail for the right lateral rectus. It can be seen that there is more activity in the greater amounts of abduction.



Figs. 31 and 32 (Magee). Human lateral rectus response during rotation of the head to the right (fig. 31) and to the left (fig. 32) while fixating zero degrees. There appears to be more motor unit firing during head rotation left than to the right. Gain 30 μv./in.; signal 250 cps., film speed 250 mm./sec.



Figs. 33-38 (Magee). (Figs. 33-37). Human left lateral rectus. Gain 100 μv./in., film speed 250 mm./sec. The records represent isometric abduction in the zero, 10, 20, 30, and 40 degrees positions, respectively. (Fig. 38). Human left lateral rectus. Gain, film speed as in Figures 33-37. See text. Read tracing from right to left.

An experiment was done on the left lateral rectus whereby the eyes were fixed binocularly in the primary position, then the left eye was abducted quickly about 15 degrees and then quickly returned to the primary position. These were repeated quite a few times and the pattern picked up was fairly characteristic. Here the low power of the preamplifier was used. Figure 38 shows this pattern. With the eyes fixing zero degrees the record shows activity typical of this, then there is marked variation in the isoelectric line. Much of the activity is lost here because of the low power of the preamplifier, but, then again, the record would not appear intact if high power of the preamplifier were used. After the marked variation in the isoelectric line (probably simultaneous with isotonic contraction of the left lateral rectus muscle) there is only mild waviness of the isoelectric line possibly related to fixation momentarily in abduction. Following this, there is what might well be inhibition possibly related to relaxation of the left lateral rectus during resumption of fixation in the zero-degree position. The initial type of activity first seen, then ensues.

Summary

1. A method of recording action potentials from the human lateral rectus eye muscle in situ is presented.

2. As in skeletal muscle the physiologic unit of contraction is the motor unit. The amount of contraction is regulated by the amount of motor unit firing at a given time; other factors probably operate but this study does not establish them.

3. The limited evidence presented would indicate that the motor unit of human eye muscle tissue is about 15 μv. to several times this in amplitude and about a millisecond in duration.

4. If motor unit firing is present in the lateral rectus while the subject gazes at infinity (a kind of "at rest" state) it is at a minimum.

5. The electromyogram of the lateral rectus during fixation straight ahead shows a definite amount of motor unit firing.

6. When the lateral rectus is in the abducted state, the amount of motor unit firing is roughly proportional to the amount of abduction. When in the adducted state, the amount of motor unit firing is roughly the same for nearly all amounts of adduction.

7. When the eye is in the depressed or elevated position, lateral rectus activity appears similar to that in eyes straight. During movement (isotonic) of the eye up or down in the zero-degree meridian the lateral rectus shows increased activity, probably a steadying action although other effects are possible.

8. Convergence and divergence as depicted in the electromyogram of the lateral rectus are oscillating movements of the globe with alternate contraction and relaxation of the muscle till the point of stabilization is reached.

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Among other articles which have appeared since preparation of this paper is the one by Moldaver and Breinen: Arch. Ophth., **54**:200-210 (Aug.) 1955.

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OCULAR OBSERVATIONS ON THE DIABETIC PATIENT*

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In the summer of 1946 and again in 1952, a survey of the ocular status of 286 children at Camp Nyda (a camp run for diabetic children by the New York Diabetes Association) was undertaken to see what percentage of the children, with no symptoms referable to their eyes, showed any eye pathology. Statistical data can be confusing, when compared with the findings of other observers, if the conditions under which the examinations are made are not similar. For instance, in my private practice, over 50 percent of the cases seen in juvenile diabetics showed pathologic ocular conditions. Here we realize the cases were referred as much for visual complaints as for routine periodic examinations.

At Camp Nyda, on the other hand, all the children of the camp were examined regardless of the fact that they had no symptoms referable to their eyes. Examination of the lens and the fundus can be inaccurate if not done through a well-dilated pupil, as the entire fundus studied systematically may show only one small pinpoint hemorrhage or microaneurysmal dilation. For this reason all the children were examined under one-percent paredrine hydrochloride and three-percent homatropine hydrobromide. A survey of this type has the drawback that only one examination can be made and no follow-ups are recorded. Still, it is interesting to note the spot findings. Complete physical

* Read before the New York Diabetes Association, May 19, 1955.

examinations including a urinalysis had been done, so that we could state that at the time of the eye examination six children showed pathologic fundus findings in the form of pinpoint hemorrhages or microaneurysmal dilations and two of these children showed albumin; two had cataractous changes and one albumin (table 1).

Cohn,[1] Lettine, Gitman, and Greenblatt, in a survey of 177 diabetic children at Camp Nyda, found that better than 50 percent showed an elevation of the serum beta lipoproteins of the Sf 12-20 and 20-100 classes.

TABLE 1

OCULAR STATUS OF JUVENILE DIABETICS*

	Camp Nyda†	Waite and Beetham
Under 10 years	186	130
10–21 years	398	464
Hemorrhages or Microaneurysmal Dilations	9	4
% Fundi	1.5%	0.6%
Cataracts	4(0.69%)	

* Two surveys at Camp Nyda (six repeats) made in 1946 and 1952 compared with the series by Waite and Beetham published in the *New England Journal of Medicine*, 212: 367, 429, 1935.

† All numbers refer to number of eyes examined not number of children.

A comparable normal group of children showed 10 percent elevations of these classes of molecules.

Interest and speculation in the project was stimulated by the following statements in the literature. Duke-Elder[2] states that patients under 40 years of age with diabetic retinopathy are exceptional. The youngest one, a 22-year-old patient, to whom he refers is the case of Adams.[3] Moore,[4] in 1925, concluded that diabetic retinitis does not occur under the age of 35 years. Waite and Beetham,[5] in 1935, studied 297 juvenile diabetics under the age of 20 years. They found no hemorrhages under the age of 10 years and in the 10 to 19-year-old group they constituted 0.8 percent.

In regard to lens changes, Waite and Beetham found flocculi cataract in four percent of their juvenile diabetics. In half of the cases in which diabetic cataract occurred the diabetes was poorly controlled.

Iannaccone and Kornerup[6] determined the plasma lipids in 100 diabetics and found the average values to be greater in the 41 with retinopathy than they were in the 59 without retinopathy.

In the two cases that showed cataract in the present series the patients were both 15 years of age, one a known diabetic of three years' duration and the other of seven years. Both showed typical subcapsular opacities. Dolger[7] has stated that 50 percent of his patients with retinal hemorrhage of diabetic origin have albuminuria. The significance of the eye findings is thus emphasized particularly when we note that the cause of albumin in the urine is changes in the kidney comparable to those observed in the retina.

In most cases we are ignorant as to why the vascular system is affected and therefore we do not know why we have retinal and vitreous hemorrhages. It is true we can alter capillary fragility by the administration of medicaments such as rutin and vitamin P, but we have all seen hemorrhages occur when the capillary fragility was normal. One should, however, bear in mind that hypoglycemic reactions even though of minor symptomatology can and do produce ocular hemorrhages as well as cerebral hemorrhages in diabetic patients with retinal vascular diseases.

In some cases the so-called "brittle diabetes" cannot be controlled well enough to obviate these reactions altogether, but in other cases it cannot be too strongly emphasized that, from the eye standpoint, a blood sugar of 140 mg. is less of a danger than a blood sugar of 60 mg., for with hypoglycemic reactions come, in many cases, retinal and vitreous hemorrhages.

A typical case is that of H. B., aged 53 years. The patient had had diabetes for nine years. For the past two years he has been on insulin (42 units N.P.H.) with occasional attacks suggestive of hypoglycemia. Vision of the right eye was 6/200 with a marked vitre-

ous hemorrhage, the vision in the left eye was 20/25 and a vitreous hemorrhage was also present here (his last blood sugar was 200 mg.). He was put on a qualitative starch-free diet by a consultant in diabetes and after a few weeks had a blood sugar of 101 mg. with no insulin. Today, 10 months later, his vision is 20/25, O.D., 20/20, O.S., and no hemorrhages. He is on 1,200 calories, mostly protein and fat, and is holding his weight at 200 lb.

In many similar cases the vitreous hemorrhages are dependent upon the associated hypoglycemia. A further suggestion in this regard is that the substitution to Lente insulin can allow reactions, if they do occur, in most cases to come about in the late afternoon and thus be detected or obviated, whereas, they might be missed if they occurred in the middle of the night.

The ophthalmologist is in a unique position to pick up early cases of diabetes. The fundus may be normal but, in an individual over the age of 21 years, if myopia increases or hyperopia decreases with no apparent cause, particularly in an individual with a family history of diabetes, a blood sugar is indicated. It is a good policy not to prescribe glasses under these circumstances until this test is done; you will be amply rewarded over the years for instituting this simple procedure.

In regard to true diabetic cataracts, an interesting observation is the rapidity in which the lens becomes mature after the cataract has once started. This is in marked contrast to congenital or hereditary cataracts. A young lady, 24 years of age, with diabetes, was led into the office with bilateral mature cataracts; from the history she had shown disturbed vision for only five months. Removal of the cataracts resulted in 20/20 vision in each eye. She only knew of her diabetes three years before the eye complication. Her last examination, March 7, 1955 (she is now aged 31 years), showed normal fundi.

Optic-nerve changes in diabetes are not common. Retrobulbar neuritis due to diabetes, which does occur, is seen so infrequently that many observers feel it is worthy of reporting. A little girl, C. B., aged 11 years, seen at Camp Nyda through the courtesy of Dr. Winifred Loughlin, gave a history of diabetes of eight years' duration. Her chief complaint was difficulty doing close work. She had been seen four years previously at which time she had 20/20, O.U., and normal optic discs. She now presented bitemporal pallor and cecocentral scotoma for red, O.U. Vision could only be improved to 20/30−, O.U. Her general examination disclosed an involvement of her auditory nerve. Complete neurologic studies and X-ray films, including sella turcica, were normal. It is felt that the diabetes could explain the picture. She is on vitamin-B_{12} injections.

On the day of surgery it is customary for the ophthalmic surgeon to give one-half the dosage of required insulin to prevent acidosis and yet not have to worry about the development of hypoglycemia. Taking into consideration the reduction of calorie intake on this day, this has seemed to work out well.

There was occasion to observe a patient that Dr. Mosenthal put on insulin 33 years ago. He gave a history of diabetes starting less than a year before. He is now 75 years of age. There were no visual disturbances until he was 73 years of age. Although he now has vitreous hemorrhages and a pathologic condition of the fundus, his case is encouraging in considering the efficacy of dietary control.

The most optimistic ray in the clouded sky of therapy in regard to ocular pathology is in the observation of the reversibility of much retinal pathology. Frequently we see cases that stop progressing and the hemorrhages and exudates absorb and the patient has a reasonably normal looking eye. A common denominator in these cases has not so far been found. Dubos[8] has written of the effect of tetraethyl ammonium chloride in protecting experimental animals against the vascular damage caused by Shiga toxin. He believes

that, since the drug interferes with the passage of autonomic impulses through sympathetic ganglions, the toxin has its primary action in the central nervous system. Likewise diphtheria toxin seems to interfere with the synthesis and function of the cytochrome system; alpha toxin of the Welch bacillus hydrolyzes the phospholipids of the cells that it lyzes. This seems to show that microbial toxins act as physiologic agents as well as antigenic controls. It is conceivable other chemicals eventually may be found which will alter or protect against the vascular damage brought on by diabetes.

Another perplexing problem is why the individual with uncontrolled diabetes is unable to stop the proliferation of tubercle bacilli or micrococci, whereas his resistance returns to normal after adequate insulin therapy. Bruger[9] has studied one diabetic patient with repeated infections for the possibility of a gammaglobulinemia. Studying by paper electrophoresis he found no lack of gamma globulin. As a matter of fact whereas a normal gamma globulin is 15 percent of the plasma proteins this patient had 22 percent gamma globulin.

Some have felt that trypsin hastens the absorption of hemorrhages; others that a combination of Methischol and Citrus Flavonoid Compound has beneficial effect on pathologic conditions of the retina. This has not been my experience.

In conclusion we should keep continuously alert to do all we can to help the present status of the diabetic patient with eye complications.

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THE ANTERIOR LIMITING MEMBRANE AND THE RETINAL LIGHT REFLEXES

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The anterior limiting membrane of the retina is insufficiently made use of as a reflecting surface in the practice of ophthalmoscopy. My attention was directed to this subject when, during routine examination of a 44-year-old adequately treated luetic woman with negative physical findings and negative serology, I noted a metallic sheen occupying the macular area overlying and slightly obscuring the retinal vessels. This glistening metallic reflex appeared to lie at the anterior surface of the retina. In an attempt to elucidate this phenomenon further and to study the anterior limiting membrane in all its various appearances, both normal and abnormal, it was my interest to examine the retinal reflexes of all patients of whatever age, both with and without pathologic changes.

In going over the literature one is struck by the unanimity with which all authors are agreed on the theoretic importance of the subject and at the same time by the scantiness of illustrative clinical material. One of the reasons for this may lie in the fact that disturbances of the retinal reflexes, although

routinely observed by the ophthalmologist, are roughly noted as part of a composite picture and are often not consciously related to the reflecting surface from which they arise. An analogous situation occurs during slit-lamp microscopy when some of the types of illumination are used without separating and classifying the resulting appearances.

In view of the intimate relationship which exists between the retinal reflexes and the character of the membrane from which they arise, it is considered worth while to review briefly the histology of the anterior limiting membrane. It is a "glass membrane of one to two mμ. thickness, showing in surface view the impressions of basal cones of Mueller's fibers as irregular polygonal fields."[1]

Its exact relationship to vitreous and retina has been a matter of some dispute. It seems to be, however, a definite structure, of measurable thickness, and it can be selectively stained by several methods. In contradiction to other authors who believed that the foot plates of Mueller's fibers join to form the anterior limiting membrane, Wolff,[2] by a method of selective staining, concluded that the end-processes of the fibers are inserted into and ramify upon a structurally distinct membrane. It is significant for my purposes to note his findings that the membrane is irregular on its retinal aspect but perfectly smooth toward the vitreous.

The basal cones of the Mueller's fibers are of importance in any consideration of the reflexes, particularly with respect to retinal regional differences. The inner ends of Mueller's fibers form conical or bulbous swellings in the nerve-fiber layer and become juxtaposed at the inner face of the retina.[3] In the central area of the retina, the expansions are shredlike rather than conical or bulbous. In the slope of the fovea their position is slanting rather than perpendicular to the face of the retina and the slanting ends become almost parallel with the inner surface before inserting themselves into the anterior limiting membrane.

Friedenwald,[4] using the Hotchkiss stain, found that the anterior limiting membrane, as well as the basement membrane surrounding the endothelium of the retinal vascular tree, stained brilliantly as a result of their carbohydrate content. He stated that the stainability of the basement membrane diminished with the age of the individual.

From the standpoint of the optical principles involved it must be remembered that the anterior limiting membrane presents a relatively smooth surface between two transparent media, the vitreous and the retina, the former having a lower index of refraction than the latter. Since the surface is not an ideally smooth one, not all the light is "regularly" reflected backward. Some of the light undergoes "diffuse" reflection on striking minute irregularities. In addition, minute irregularities in the surface cause formation of secondary images in the regularly reflected beam and this is called "specular reflex of the reflection surface."[5]

The retinal reflexes occurring clinically as regularly reflected light, or as specular reflex, can only exist when the incident light strikes the retina at an angle such that the reflected ray returns to the eye of the observer. For this to occur the angle of incidence and therefore the angle of reflection must necessarily be rather small. Only in the central areas of the retina does this situation exist and only here may we see the retinal reflexes. An incident ray striking the retina peripherally would upon regular reflection lose itself in the eye.

The retinal reflexes have been described by Dimmer[6] at great length, and by Ballantyne[7] and Danis[8] more recently. They deserve re-emphasis at this time since recognition of alterations of the normal reflex and its variants often enables the observer to spot otherwise easily overlooked pathologic conditions. No recent references to this subject exist as far as I know except in a recent textbook of ophthalmoscopy,[9] and the description here of pointlike reflexes, to be alluded to later, does not coincide with my own findings.

The retinal reflexes may be divided for convenience into macular and extramacular reflexes on the basis of location and quality.

In both cases there is constant movement of the reflex as the direction of the ophthalmoscope light changes with respect to the subject's eye, and with respect to minute alterations in the surface of the anterior limiting membrane. The observer can readily form an impression as to the texture and mirroring qualities of the membrane.

The macular reflex is finely granular. With movements of the ophthalmoscope light, it approaches the fovea never quite reaching it, recedes from it just beyond the limit of the anterior and superior temporal retinal vessels, and goes from side to side encircling the fovea. The impression one gets is of a superlatively smooth anterior limiting membrane.

The light reflex often bears striations produced by the underlying arcing fibers of the nerve-fiber layer. There may also be seen, but much less frequently, vertically oriented closely parallel lines between the fovea and the disc. I am unable to give an explanation for the latter phenomenon. The foveal reflex is pointlike or bloblike and its altered character or absence is recognized as an important clinical sign in pathologic conditions of the macula. It is a specular reflex of the reflecting surface of the foveal concavity.

The extramacular reflexes occur in an area of the fundus extending inferiorly, superiorly, and nasally from the disc for one to three disc diameters. The reflexes are accentuated along the hillocks produced by the retinal vessels as they issue from the disc, and are definitely anterior to the reflexes produced by the vessel walls themselves. The so-called Weiss' reflex is a linear one paralleling the nasal border of the disc and arising from a ridge made by the nerve fibers as they dip to enter the nasal side of the nerve. Elsewhere in this extramacular or peripapillary area the reflex often gives the impression of a beaten gold plate. At any rate the texture of the membrane seems to be less fine than that in the macular area.

Studding the extramacular reflex are innumerable small points of light which represent the foot-plates of the Mueller's fibers inserted into the internal limiting membrane. They are never seen in the macular area probably because of the obliquity of insertion, previously noted, of these fibers into the limiting membrane of this zone. Adalbert Fuchs[9] described them as Gunn's dots and stated they are present in the macula when they exist. In my experience they are never found in the macula, but are almost invariably present extramacularly.

These pointlike reflexes when studied more carefully are noted to be irregularly polygonal, occasionally rectangular, or dotlike. These appearances are undoubtedly altered by the presence or absence of astigmatism. Each seems to represent a regular reflection from a surface rather than a specular reflex from the conical end of the Mueller's fiber in the nerve-fiber layer. They appear to show no parallactic displacement. They are distant from each other about 0.05 mm. Occasionally their distribution follows the direction of the fibers in the nerve-fiber layer.

The brightness of the point reflexes is roughly in accord with the brightness of the extramacular reflex which they stipple, although occasionally one may be seen without the other. At times, in young people, the retinal reflex is so bright that it is only with difficulty that the pointlike reflexes are seen.

The points of light are sometimes replaced, particularly one or more disc diameters from the disc, and especially in the nasal direction, by points of darkness, or by points of darkness sharply outlined by a partial ring of light. These appearances suggest that the surfaces of the foot-plates of Mueller's fibers not always at the same inclination or at the same level as the anterior limiting membrane.

Rarely another type of point reflex is seen which seems to be due to an entirely incidental irregularity of the retinal surface of no particular significance.

In general, it should be said that a very large normal variation exists as to brilliance or indeed presence or absence of reflexes. However, the observer familiar with reflexes

will recognize the limits of the normal, keeping in mind the possible individual variations, and the factors of age and refractive error.

First as to age:

It has already been suggested that a chemical change probably occurs in the anterior limiting membrane with increasing age. That this is accompanied by a physical alteration in the mirroring quality of the membrane can be considered probable. Dimmer's suggestion is a most interesting one, namely that the refractive index of the retina diminishes, or that of the vitreous increases with age, thus producing diminution in the quantity of regularly reflected light. Whatever the explanation, at the age of 15 to 40 years, the reflexes have often to a great extent disappeared, but the individual variation is still a large one.

In later years, past the age of 50 or 60 years, fixed reflexes are noted in the paramacular areas. These vary in size from twice the diameter of a retinal vessel to a size encompassing a part or the whole of the macula. They seem to represent a local senescence in the anterior limiting membrane, and often occur in an otherwise perfectly normal eye. I have called them fixed reflexes because they constantly recur at the same area when studied with a moving ophthalmoscope and have been seen in the same location without change for three years or more.

In addition to the factor of age, the refractive status is well recognized as influencing the brightness of the reflexes. The hyperopic eye shows much brighter reflexes than its myopic opposite number. No particular reason for this has been discussed in the literature. It may well be that the tenseness of the anterior limiting membrane and thereby its physical characteristics as a reflecting surface are more favorable in the hyperope than in the myope.

Other factors such as retinal or choroidal pigmentation have not seemed to influence the character or the brightness of the reflex except in supplying a more or less favorable background for observation. The use of red-free light presents no special advantage in the study of the reflex.

In the present state of our knowledge it is possible to remark only that the reflex is normal or abnormal. A more definitive diagnosis has to be based on other clinical grounds. However, the obviously pathologic reflex calls attention to an alteration in the chemical or physical state of the anterior limiting membrane produced by inflammation, edema, neoplasm, tenseness, or hemorrhage.

Bernard Samuels and Adalbert Fuchs[10] state that "In pathologic conditions a row of cells similar to a layer of endothelium may be found on the surface of the limitans interna. The origin of these is not known. It is similar to the layer on the surface of the iris in chronic inflammation."

Duke-Elder[11] describes new formation in the form of a membrane overlying the retina occurring after mild chronic inflammation.

Bertha Klien[12] in a case of infantile amaurotic idiocy reported groups of round cells applied to the inner retinal surface and suspended in the posterior vitreous. She considered this a low-grade inflammatory reaction caused by "prolonged contact with toxic products of tissue degeneration."

Samuels and Fuchs[10] further described detachments of the limitans interna produced by hemorrhage or edema. They recognized the intimate connection existing between the foot-plates of Mueller's fibers and the anterior limiting membrane, but postulated a difference in the firmness of this attachment from individual to individual. On the other hand, in view of the absence of the point reflexes in the macular area and in view of the histologic regional differences as described earlier in the paper, it may be that the attachment is much less firm in the macula than elsewhere.

A frequent ophthalmoscopic finding is dark striations running radially from a pathologic focus. These striations are apparently the result of wrinkles or folds in the anterior limiting membrane produced by lines of tension.

It is obvious, then, that a wide range of ocular conditions—hemorrhage, neoplasm, edema, inflammation—may produce changes in the mirroring quality of the membrane, in its position with respect to the rest of the retinal surface, in the translucency of the adjacent vitreous and retina, and in the tenseness of the membrane, thereby producing changes in the retinal reflexes.

Of course, many questions remain unanswered. Does the index of refraction of the vitreous and of the retina change with age and, if so, how do these changes affect the retinal reflexes? What are the chemical and physical causes of the fixed paramacular reflexes of the senescent eye? Are there vitreous adhesions at the sites of these reflexes? What relationships can be developed between abnormal retinal reflexes in general and the histologic structure of the anterior limiting membrane?

SUMMARY

1. The histology of the anterior limiting membrane was reviewed.

2. Macular and extramacular light reflexes were described, special emphasis being placed upon the regional differences and upon the factors of age and refractive error.

3. Attention was called to the pathologic conditions responsible for alterations in the retinal reflexes.

4. The point was made that knowledge of the retinal reflexes and their normal variations allows the observer to spot otherwise readily overlooked pathologic processes, old and new.

1777 Grand Concourse (53).

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MYOPIA CAUSED BY PREMATURITY*

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Passing reference to myopia, occurring after prematurity, has been made in the literature. Few direct references to this type of myopia are available. Most of the comments have occurred in literature concerning the syndrome of retrolental fibroplasia.

Obviously, the vision, following retrolental fibroplasia of grades 1 or 2, should be better than after more serious grades. The cases I am reporting are all children with relatively good vision.

Based on the classification of cicatrical retrolental fibroplasia,[12] established by the joint committee of the National Society for the Prevention of Blindness, useful vision may follow stages 1, 2, and 3.

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TABLE 1

BIRTH WEIGHT AND MYOPIA

Case	Birth Weight	Year	Myopia	Year	Latest Vision
1	4 lb. 3 oz.	1946	R −7.00 −2.00×90 L −6.00	1953	R 20/20 L 20/80
2	Born elsewhere	1940	R −16.00 L −14.00	1954	R 20/20 L 20/50
3	4 lb. 6 oz.	1948	R −9.00 −50×180 L −8.00 −50×180	1954	R 20/20 L 20/20
4	5 lb. 0 oz.	1948	R −50 −1.00×105 L −9.00 −1.00×105	1954	R 20/60 L 20/200
5	Born elsewhere	1948	R −4.00 −3.50×30 L −4.50 −2.50×180	1954 (Telescopics)	R 20/30 L 20/40
6	5 lb. 1 oz.	1937	R −13.00 −1.50×165 L −13.00 −1.00×15		R 20/30 L 20/25
7	4 lb. 1 oz.	1939	R −5.25 −2.00×5 L −1.25 −1.00×160	1954	R 20/20 L 20/20

These children all had birth weight of four to five pounds, and, comparing these cases to Reese's 99 of the same birth weight, he noted eight in stage 1 and 2 and 20 in stage 3 and 59 in stage 5 of the cicatricial retrolental disease. It may, therefore, be assumed that these cases of mine represent approximately the usable eyes from 100 premature infants, weighing between four and five pounds, birth years 1937 to 1953.

The cases which I am reporting are, therefore, in this group of healed retrolental fibroplasia.

My cases are all those in which prematurity was a large factor during the early weeks following the birth of the child. I did not see these cases during oxygen treatment, although I have attempted to verify the amount of oxygen received in each case.

In view of the certainty of premature birth, and the fact that each of these cases shows a specific syndrome, I am suggesting that they may represent a type of myopia which was caused by retrolental fibroplasia, and I shall outline the details of this type of myopia, as I have seen it in my patients, some of whom I have followed for 14 years (table 1).

Reese[7] states that the refractive error in these cases is usually four to eight diopters. He tabulated the vision in 15 cases and associated it with a definite stage of cicatrical retrolental disease. Ten of his cases were classed as stage 3 and a temporal retinal lesion was most frequently seen. The vision in his cases varied between 10/20 and 20/20.

King,[8] in discussing his series of 238 cases, had 17 in which vision was better than 20/200.

Reese states that 62 percent of his large series showed grade 5 cicatricial changes; however, he also believes that a small number of eyes in his series were entirely normal after active retrolental fibroplasia, even with inspection under general anesthesia.

When speaking of myopia, which occurs after retrolental fibroplasia, certain factors are seen to occur regularly. This type of myopia is quite different from the well-known developmental type of myopia which occurs around the age of puberty and is referred to as "axial myopia." it is also different from the severe type of progressive myopia called "malignant myopia," but the myopia of prematurity is extensive enough to resemble malignant myopia more than the benign forms of myopia.

In my experience, myopia in children who were born prematurely frequently is seen as a severe grade of myopia, perhaps 10 or 11 diopters, when first discovered at an early age. Lesser grades of early myopia may improve to nearly normal before diagnosis is made. The condition is not rapidly pro-

gressive and, if discovered at the age of one or two years, often does not progress for the next decade.

This form of myopia may be associated with either optic atrophy or even mental retardation as the result of birth injury or oxygen damage to the brain. Paralytic changes in some of the extraocular muscles often lead to strabismus. Operation on the muscles is frequently required because no other treatment will improve the cosmetic appearance.

The best corrected visual acuity is often less than 20/20.

Case histories

Case 1

Betty A. came to the ophthalmologist at the age of one year. She was prematurely born, weighing four and one-half pounds. Her eyes had turned in since the age of six months. She had nystagmus. Retinoscopy showed: (cycloplegic) R.E., −7.0D. sph. ⌒ −2.0D. cyl. ax. 90°; L.E., −6.0D. sph.

Glasses were prescribed and the child was followed from 1947 to 1954. Her myopia at that time measured: R.E., −9.0D. sph. ⌒ −1.0D. cyl. ax. 180°; L.E., −9.0D. sph. ⌒ −1.0D. cyl. ax. 180°. Her convergent strabismus was cured spontaneously without surgery. Her nystagmus decreased progressively.

Her best vision was: R.E., 20/20; L.E., 20/80. The child was able to do first-grade work satisfactorily.

Examination of the retina showed that there were very tortuous vessels, and a few vitreous opacities without any choroidal retraction. There were large areas of choroidal atrophy.

It is to be noted that in seven years there was very little progress in the myopia. It is postulated that this myopia is the result of a reversible form of retrolental fibroplasia.

Case 2

Eugene O. came to the ophthalmologist at the age of six years. He was a prematurely born baby with a convergent strabismus. His first retinoscopy showed: R.E., −15.0D. sph.; L.E., −15.0D. sph.

Between 1946 and 1954, his myopia did not change appreciably. In 1950, retinoscopy revealed: R.E., −16.0D. sph.; L.E., −14.0D. sph.

This patient was very slow to develop mentally and his eyes required surgery in order to be straightened. His vision was slow to develop and it was only in 1954 that I was able to get 20/30 in the right eye and 20/50 in the left. This child also had nystagmus and did not do well in school. At the age of 12 years, he was in third grade.

His retina showed opacities in the vitreous together with retraction of the choroid. He also showed degenerative areas in the periphery.

In view of the prematurity and the myopia, which did not progress appreciably over a period of eight years, this case is believed to belong to a particular type of myopia which is the result of a reversible form of retrolental fibroplasia.

Case 3

Thomas W. was prematurely born at seven months and weighed four pounds. He came to the ophthalmologist at the age of two years. Retinoscopy revealed: R.E., −9.0D. sph. ⌒ −0.5D. cyl. ax. 180°; L.E., −8.0D. sph. ⌒ −0.5D. cyl. ax. 180°. In four years, there was no change in the retinoscopy.

This patient had straight eyes and, in 1954, 20/20 vision in each eye. His retina showed highly myopic signs with pigment granules spread thinly over the posterior pole. The periphery showed less scarring than the other cases.

In view of the prematurity and high myopia, without progress over four years, this case is believed to represent part of the syndrome of reversible retrolental fibroplasia.

Case 4

Peter P. was born prematurely at the eighth month and weighed five pounds. He

consulted the ophthalmologist at the age of five years. Retinoscopy revealed: R.E., −0.50D. sph. ⌒ −1.0D. cyl. ax. 90°; L.E., −9.0D. sph. ⌒ −1.0D. cyl. ax. 105°. This child is believed to represent a later and less malignant form of the syndrome of reversible retrolental fibroplasia because he was born nearly at term and only one eye was affected. His parents' eyes were entirely normal.

His vision was correctible, at the age of five years, to 20/60 in the right eye and less than that in the left eye.

Case 5

Lee F. was referred to me at the age of six years, having been born prematurely and having spent several months in the hospital because of this. Vision, without glasses, was 10/60 each eye. Retinoscopy showed (cycloplegic): R.E., −4.0D. sph. ⌒ −3.5D. cyl. ax. 30°; L.E., −4.0D. sph. ⌒ −2.5D. cyl. ax. 180°. Vision was approximately 20/60.

Telescopic spectacles, ×2.2 magnification over the retinoscopy equalled: L.E., 20/30. R.E., 20/40.

Examination of the fundus revealed a thin retina with many evidences of high myopia, including several small vitreous opacities. My diagnosis was myopia associated with prematurity. Glasses were prescribed.

Case 6

George C. was examined at the age of 17 years. There was no history of myopia in his family. His mother reported that he weighed five pounds, one ounce at premature birth; this weight is bordering on normal, even though he was delivered at eight months. He has had glasses since the age of six years, and his myopia has not progressed during the time I have followed him.

Ophthalmoscopically, his media are clear and the macula normal. There is pigment scattered through the retina, but no evidence of choroidal or other degeneration. Retinoscopy reveals: R.E., −13.0D. sph. ⌒ −1.5D. cyl. ax. 165°; L.E., −13.0D. sph. ⌒ −1.0D. cyl. ax. 15°.

Case 7

Robert K. was first examined at the age of two years. He had been born prematurely at seven months, weighing four pounds, one ounce, and he was kept in an incubator for six weeks. He had, when first seen, a convergent strabismus, which was operated upon in 1942, at the age of three years.

His first retinoscopy, in 1942, was: R.E., −4.0D. sph.; L.E., −0.5D. sph. His last retinoscopy, on October 11, 1954, at the age of 15 years, was: R.E., −5.25D. sph. ⌒ −2.0D. cyl. ax. 5°; L.E., −1.25D. sph. ⌒ −1.0D. cyl. ax. 160°. Vision with glasses was 20/20, each eye, and the eyes were straight.

Examination of the fundus has never shown any vitreous opacities or any retinal lesions.

This patient is included to show, over a 14-year period, the relative lack of progress in these cases.

Etiology

In this type of myopia, the history of premature birth, often requiring prolonged hospitalization of the baby, is always obtained.

Based on the work of Szewczyk[14] and Ryan[18] (1952) who first focused attention on the role of oxygen in the premature, and upon the exhaustive reviews of Zacharias,[11] Maumenee[20] and Wagener,[21] this type of myopia is believed to be one form of regression from the vascular changes induced in premature eyes by the administration of oxygen or its sudden withdrawal.

In the classification of cicatrical retrolental fibroplasia, these cases would fall under the mildest groups, namely, groups I to III. They are the cases in which a retinal vascular abnormality occurred early in life but regressed sufficiently to avoid the formation of vitreous bands or detached retina.

Sometimes vitreous hemorrhages have occurred, as may be seen from residual

vitreous opacities and the areas of peripheral atrophy which are the scars resulting from the initial lesion. It is postulated that the vascular crisis, caused by oxygen and its sudden withdrawal in the premature eye, affects the vasculature of the choroid in such a way as to allow the development of severe myopia.

Very little is known about the effect of oxygen on the choroid of prematures. Manschot[15] assumes that the choroid has a higher oxygen tension than the vitreous because of its vascularity, and cites the work of Michaelson[22] and Campbell,[22] who demonstrated that capillaries grow in the direction of the lowest oxygen tension.

While the relatively higher oxygen tension of the choroid may keep it free of the most damaging changes in retrolental fibroplasia, it is presumable that some vascular effect of oxygen administration is present in the choroid.

Ashton[24] showed that high concentrations of oxygen obliterate the retinal vessels of the premature kitten. Whether the myopia of prematurity comes about because of the obliteration of choroidal capillaries in the period of oxygen administration or because of the suspension of growth of the vascular tree in the choroid, while the rest of the eye grows, must be determined later. By whatever cause, these children seem to be more severely affected in their early years and, instead of progressing to more serious stages, seem to improve with normal growth, in contrast to the behavior of progressive myopia.

Manschot emphasizes a point which has also been noted by Dr. R. Tenant in our laboratory, namely, that these premature children frequently suffer from interstitial pneumonia, which increases the hypoxia and adversely affects the eye.

Differential diagnosis

In my practice, I see two well-known types of myopia. The most common is *axial myopia* or developmental myopia, the other type is *malignant myopia* (table 2).

Other varieties include the late myopia due to lens swelling, sometimes referred to as the development of "second-sight," by lay persons, who become able to read again without glasses.

Axial myopia develops in hyperopic eyes that lose their hyperopia during growth. The onset of myopia follows loss of the usual hyperopia and begins in the teen-age period, increases slightly year by year, the increase being more in the years of greatest growth. This type of myopia gradually stops increasing when full growth is attained, and when extensive near work, usually study, is no longer required. The average extent of this myopia is between two and four diopters. It is usually not associated with any ocular pathology, such as detached retina, and persons having it generally go through life with no ocular problems[1] that may be attributed to

TABLE 2

Myopia

	Axial	Malignant	Premature
Onset age	10–15 years	Early 3–6	0–1
Progress	0.5 diopters, a year or less, for 10 years	Often 2–3 diopters a year, lifetime	None, after birth
Visual acuity with glasses	Normal	Reduced, increasingly	Improves to normal
Complications	None	Detachments Choroiditis Cataracts	Strabismus Nystagmus Microphthalmia
Dioptric extent	−1.00 to −5.00 diopters	−6.00 to −40.00 diopters	−6.00 to −12.00 diopters
Prognosis	Good	Guarded	Good

TABLE 3

RÉSUMÉ OF PREMATURE HISTORY

Case	Birth Weight	Duration, Oxygen	Year	Type Delivery	Comment
1	4 lb. 3 oz.	16 days	1946	Spontaneous, 8 months	
2	Born elsewhere		1940		
3	4 lb. 6 oz.	20 days	1948	Caesarian, 7½ months	
4	5 lb. 0 oz.	?14 days?	1948	Spontaneous, 8 months	Eyes (in hospital) examined, RLF
5	Born elsewhere	several months in hospital	1948		
6	5 lb. 1 oz.	?	1937	8 months	
7	4 lb. 1 oz.	6 wk.	1939	7 months	

My diagnosis is based on the history of prematurity and the findings of myopia; and this history of prematurity was either proven by the hospital record or believed true because of the reliability of the informant. In 1946 and 1948, the suspicion of oxygen had not become a factor in the premature nursery, so it is natural to have inadequate records of oxygen usage.

Only one of the cases was examined at birth by an ophthalmologist (Dr. A. C. Unsworth), and he noted a pupillary membrane across the eye which later showed the most severe changes of myopia, although the membrane had disappeared by the age of five years. Seven days after birth, he noted "retinal pallor in the periphery and peripheral elevation."

the presence of myopia.

Malignant myopia, so called because it often continues to progress for the life of the patient, and may reach −20.0D. or −30.0D. in extent, is accompanied by retinal degeneration and choroidal atrophy so that visual acuity is often severely reduced.

Malignant myopia is believed to be an hereditary condition; it may also be adversely affected by malnutrition, hypocalcemia, and avitaminosis.[2-4]

Myopia of prematures is always found in children who were born prematurely and is usually discovered at the age of one or two years. There is no hereditary factor. Often, both parents have normal or hyperopic eyes.

The myopia of prematures is usually advanced when first discovered, sometimes to −8.0D. or −12.0D. (table 3).

This type of myopia usually does not progress but frequently improves, both in extent and in visual acuity, as the child develops.

It is associated with different types of complications, such as strabismus, nystagmus, and optic atrophy, rather than retinal atrophy or choroidal degeneration.

Prognosis

The myopia of prematures seems, on the basis of these few cases and on the basis of its cause, to have a good prognosis.

Once the premature vascular reaction to hypoxia has been overcome, the eye, although severely affected, proceeds to strengthen and carry on to the point where it eventually functions as well as eyes which were not severely damaged in early life.

The recovery process takes five years at least, in the course of which muscle surgery is often required, but the final result is adequate for normal living. Often, after recovery, these eyes show no evidence of their severe period of shock. The diagnosis must be made in early life. While there is no reason to suppose these eyes are not likely to change into other types of myopia, I have no evidence that any of my cases changed from one type of myopia to another.

Given reasonably good inheritance, these babies overcome the deficiency that occurred during their premature age.

Management

I have been satisfied to treat these babies with glasses early in life and to add any supportive measures available. When I find 10 diopters of myopia in a child one or two years of age, I usually prescribed only half the total correction for three to six months; later on, increase to the full correction required.

As the visual acuity improves, I have noted

in some cases, a reduction in the correction required. Some of these cases require sight-saving classes in early grades, but usually improve enough to carry on with normal school print by the fourth grade.

Those cases not improving have additional complicating factors, such as brain injury from birth trauma, or congenital cerebral deficiency, or nystagmus. Of these cases, a few are benefited by telescopic spectacles.

Conclusion

A new form myopia has been described. Associated with prematurity, it is unusual in that it does not progress after its initial occurrence. This type of myopia does not fall in the category of other and more common types of myopia, described by previous authors (Tschernig, Prangen, Burton, Stansbury, and others).

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EFFECT OF TRAUMA ON THE PRODUCTION OF EXPERIMENTAL UVEITIS*

A REPORT OF PRELIMINARY STUDIES

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INTRODUCTION

Experimental uveitis may be induced in animals with a variety of antigens.[1,2] One method is by a single injection of such agents as certain of the gram-negative bacilli.[3] However, this type of ocular response, in all probability, is a direct toxic effect of these antigens on uveal tissue. Two other procedures which will produce uveitis and are considered to be true hypersensitivities are:

1. General sensitization of the animal and subsequent administration of the shock dose intraocularly.

2. Sensitization of the animal intraocularly and subsequent administration of the homologous antigen extraocularly (usually intravenously). Both methods require the direct injection of the nonliving antigen into the globe. The present work, therefore, was instigated to determine whether specific ocular reactions could be elicited by nonperforating trauma to the eyes of sensitized rabbits and nonsensitized rabbits. With such a procedure the direct intraocular injections of the antigens could be avoided.

METHODS AND MATERIALS

Normal albino rabbits of varying weights and both sexes were used throughout this study.

These animals were generally sensitized to 1:10 commercial bovine albumin† (0.5 ml. intravenous and 0.5 ml. intramuscular injections every three days for a period of 15 days). After the course of sensitization, the right eyes of a group of rabbits were traumatized by the method described below. The nonperforating injury was incurred approximately one to two mm. from the limbus at the 12-o'clock meridian. Seven to 10 days later (a period of time which was found to be sufficient for the effects of the trauma to subside) the animals were given 3.0 ml. of the homologous antigen into the marginal ear veins (table 1).

Controls consisted of animals which were not sensitized but traumatized and shocked; those which were sensitized and shocked but not traumatized; and those which were shocked but not traumatized nor sensitized.

The trauma was performed as follows: The animals were anesthetized locally with four-percent cocaine. The instrument for trauma was essentially a modified "sling shot" composed of a blunt-end rod which was attached to a stage, the driving force was obtained by a No. 64 Plymouth rubber band. A new band was used for each rabbit. In order to obtain some uniformity for each trauma, a scale was drawn on the stage so that the rod might be withdrawn (prior to release) to the same point each time.

The agar diffusion method for determining the presence of antibodies in the aqueous humor was carried out as follows:

Paracenteses were performed on five sensitized animals seven days after the trauma. These animals did not receive the shock dose of albumin. The aqueous humor was concentrated by placing three drops on a small filter paper disc (12.7 mm. in diameter) and desiccating under a high vacuum pump. This

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† Bovine albumin (30-percent solution) was obtained from The Armour Laboratories. Each milliliter contained 0.3 gm. of albumin prepared from bovine plasma.

TABLE 1

OCULAR RESPONSES OF ANIMALS SENSITIZED, TRAUMATIZED, AND SHOCKED WITH BEEF ALBUMIN

Animal No.	General Sensitivity to Albumin	Trauma	Antigen for Shock (intravenous)	Ocular Condition Immediately before Shock		Reactions after Shock					
						2–4 hours		24 hours		48 hours	
				O.D.	O.S.	O.D.	O.S.	O.D.	O.S.	O.D.	O.S.
207	X	X	X	0	0	+++	0	+	0	0	0
202*	X	X	X	++	0	++++	0	++++	0	+++	0
201	X	X	X	0	0	++	+	+	0	0	0
224*	X	X	X	0	0	++	+	+	0	+	0
223*	X	X	X	0	0	+	0	++	0	0	0
208 (Control)	0	X	X	0	0	0	0	0	0	0	0
205 (Control)	0	X	X	0	0	0	0	0	0	0	0
203 (Control)	0	X	X	0	0	0	0	0	0	0	0
227 (Control)	0	X	X	0	0	0	0	0	0	0	0
204 (Control)	0	0	X	0	0	0	0	0	0	0	0
211 (Control)	X	0	X	0	0	0	0	0	0	0	0

X: Treated
0: Not treated
* Indicates intraocular hemorrhage following trauma

procedure was then repeated. Three drops of antigen (1:10 ml. of bovine albumin) were similarly placed on a disc. The discs were separated by a distance of 0.7 of a centimeter in a petri dish containing 0.8-percent agar prepared in buffered physiologic saline (pH 7.4). The covered plates were incubated at 4°C. The readings were made at frequent intervals for two weeks. A positive test as indicated by a line of precipitate between the antibody and antigen discs was observed after two days in incubation (table 2).

FINDINGS

As may be noted in Table 1, the five sensitized animals which were subsequently traumatized and shocked developed ocular inflammation in those eyes which had been subjected to nonperforating injury. The reaction may be considered an immediate or anaphylactic type since the ocular response appeared macroscopically within two hours after the shock dose was administered intravenously. The fellow eyes (nontraumatized) demonstrated minimal if any ocular inflammation.

The reactions were characterized by conjunctival hyperemia, circumcorneal injection, and iritis. The control animals did not demonstrate ocular inflammation, indicating that both general sensitization and trauma were necessary factors in the production of this type of uveitis.

The five sensitized rabbits (table 2) from which aqueous was withdrawn seven days after the trauma demonstrated antibodies in the right eyes (traumatized) and absence of these immune bodies in the left (untraumatized). Unfortunately the greater portion of the two samples of aqueous which were re-

TABLE 2

DETERMINATION OF ANTIBODIES IN AQUEOUS OF RABBITS SENSITIZED AND SUBSEQUENTLY TRAUMATIZED

Animal No.	General Sensitivity to Albumin	Trauma	Antigen for Shock (intravenous)	Presence of Antibodies 7 Days After Trauma	
				O.D.	O.S.
270	X	X	0	SI	0
214*	X	X	0	+	0
103	X	X	0	SI	0
120*	X	X	0	+	0
123	X	X	0	+	0

SI: Sample inadequate for concentration.
X: Treated
0: Not treated
* Indicates intraocular hemorrhage following trauma

moved from the experimental eyes was lost due to a laboratory accident. Consequently, the quantity of aqueous was inadequate for concentration of the gamma globulin. The aqueous from the traumatized eyes differed qualitatively from those obtained from the fellow eyes as evidenced by increase in fibrinous material in the former. Under these conditions, this finding would indicate that plasmoid aqueous may remain in the anterior chamber at least seven days after trauma has been inflicted.

Discussion

Traumatic uveitis in humans is a distinct clinical entity yet the mechanism of such an inflammatory reaction following a nonperforating injury is not clearly understood.

Rones and Wilder[4] state that "unquestionably the injury and destruction of tissues can liberate toxic substances which induce an inflammatory response. In the same way, intraocular hemorrhage can act as an exciting agent. There are two less likely factors which nevertheless have their proponents; namely, reflex nerve irritation and allergic sensitivity to dispersed uveal pigment or lens proteins."

The work presented here, although of a preliminary nature, would indicate that possibly hypersensitiveness to foreign proteins might also play a role in traumatic uveitis. Of the animals tested, no iritis could be produced by trauma followed by shock *unless* they had been sensitized prior to injury. Controls ruled out the possibility of ocular responses occurring in animals which were generally sensitized and were subsequently shocked by the intravenous route. However, previous experiments have shown that, if the eye is sensiitzed or shocked directly, such iritides could very possibly occur.

It is tempting to speculate as to the mechanism involved in these experimental inflammations. Trauma seemingly acts as the trigger mechanism by modifying the blood aqueous barriers and thus allowing antigen and/or antibodies to enter the anterior chamber. That circulating immune bodies were present was demonstrated by the agar diffusion techniques. The inflammatory reaction in all probability resulted from a classic antibody-antigen combination with the release of histamine or histaminelike substances by the injured cells.

Studies are now in progress to determine whether antigens which have been shown to be capable of sensitizing uveal tissues can also elicit this type of experimental uveitis.

Also, presently we are comparing the agar diffusion method for determining antibodies in the aqueous with the capillary tube method[5] to determine which procedure is the most sensitive.

Summary

1. Specific ocular reactions could be elicited in rabbits sensitized with beef albumin and subsequently traumatized and shocked.

2. Control animals did not demonstrate experimental uveitis which would indicate that both general sensitization and trauma were necessary factors.

3. Antibodies were demonstrated by the agar diffusion method in the experimental eyes (traumatized). No immune bodies could be found in the fellow eyes (nontraumatized).

4. A possible mechanism involved in experimental traumatic uveitis is discussed.

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NOTES, CASES, INSTRUMENTS

RADIOLOGIC-SLIT METHOD FOR FOREIGN-BODY LOCALIZATION*

A PRELIMINARY REPORT

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In order to establish a favorable prognosis in cases of ocular foreign bodies, it is necessary to know the nature and dimension of the foreign body, as well as its exact topography. Although the first factor usually creates little difficulty, the second may become an urgent problem when the foreign body cannot be visualized ophthalmoscopically. In spite of numerous techniques for localization, as yet no method is really simple, exact, and harmless.

There is no doubt that radiographic exploration is essential but it is well known that frontal or lateral X-ray films do not permit visualization of the contour of the globe and, for this reason, permit only suppositions as to the exact location of the foreign body. In addition, it is not safe to use X-ray films to locate bodies near Tenon's capsule.

Opaque substances may be used for references at the anterior pole but not at the posterior limits of the globe which, in the majority of cases, is where they are needed. Contrast substances (air, lipiodol, Diodrast) have been used to visualize the posterior hemisphere without much success and with much discomfort to the patient.

Sweet's technique and the stereographic method of Senná require expensive apparatus and complicated calculations, making these procedures impractical. Argañaraz's rings are inconvenient because of their arbitrary position and size. Caballero's hook-ring is dangerous and difficult to apply.

In its day, planigraphy seemed to hold promise but, after many attempts and waste of much time and X-ray films, the method proved inefficient. Disappointing also were the techniques used by the Radiology Department of the University of Uppsala.

After many trials and errors, it occurred to us to try a totally different method—that of utilizing the human eye's own sensitivity to X rays. Brandes and Dorn (1897) were the first to notice that the human eye recognized roentgen rays as a light sensation. Beloit verified these findings in 1905. Later (1938) Rushton and Sorsby employed X-ray beams to measure the axial length of the living human eye. Goldmann and Hagen (1942) and Sorsby and O'Connor (1947) also used X-ray beams to study the eye.

PRESENT TECHNIQUE

Having proved in a series of patients of subnormal mentality that the human eye is capable of appreciating narrow ribbons of roentgen rays as a luminous sensation, we focussed a vertical beam of very narrow rays in such a manner that they were dispersed gradually in an occipital to frontal direction, at the spot on the posterior limit of the globe pointed to by the patient when he received the sensation of light. At this instant, a lineal radiogram was made on a film placed vertically tangent to the posterior part of the ocular globe. Immediately and with no change in the patient's position, and without closing the first slit, but opening the second slit, a lateral, normal radiogram of the skull was taken on the same film. In this way, on the double-exposure radiogram, were obtained the normal image of the patient's skull and over it a black line, well limited and tangent to the posterior pole of the globe. The first attempts were not perfect but, after further study, a technique was evolved by which it is possible to show the tangent line in both posterior and anterior parts of the

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Fig. 1 (Junceda and Noriega). X-ray films, showing results of radiologic-slit technique. Note the superimposed line in each view. Because the patient moved, the horizontal line in the left-hand picture is somewhat higher than it should be.

globe. Although it seems unnecessary to remark, it is also possible to obtain tangent lines in any part of the superior or inferior hemisection.

In our technique, it is necessary to locate a point in space with reference to three principal planes; in case of doubt, once the lateral radiogram has been taken another exposure may be made in a frontal position. In addition the complete immobility of the patient is essential.

The details of the technique are:

1. To dark-adapt the patient, his eyes are covered with pads for 25 to 30 minutes.

2. Careful placement of the patient so that the X-ray beams are tangent to both globes.

3. Exploration of the globe in the desired area with a wide beam (slot-shaped, 1.5 mm.) so that the patient may learn to recognize the luminosity.

4. Focussing the apparatus through a one-mm. slit, gently moving the beam forward or backward, upward or downward.

5. Adjusting the film without moving the patient, a one-second exposure is made with 78 K. V. (These values may, of course, be changed according to the speed of the film and the characteristics of the apparatus.)

6. Both slits are opened and the second, shorter (0.3 second) exposure is made.

The distances used are:

1. From the tube to the film, 50 cm.

2. From the first slit to the second, 22 cm.

3. From the second slit to the skull, 2.0 cm.

The dimensions of the slit are: First slit, 3.0 cm. by 5.0 mm; second slit, 3.0 cm. by 0.5 mm.

Discussion

When a narrow slit is used at the distances designated herein, the diversion of the X rays is practicably nil. However, it should be kept in mind that the anterior border of the slit is tangent to the retina and, therefore, in the radiogram, the posterior edge is the one tangent to the globe, for the thickness of the sclera and the choroid is approximately the same as that of the slit used.

Summary

The radiologic-slit method herein described is not difficult to execute, nor does it require any special or expensive apparatus. It entails no risks for the patient, for the radiation used is less than that commonly used in neurology.

This is a preliminary communication only;

soon we hope to complete a detailed study. In our opinion, however, this technique can satisfactorily solve the problem of localization of foreign bodies.

Principado 7

CONGENITAL PARALYSIS OF SIXTH AND SEVENTH NERVES

CONGENITAL FACIAL DIPLEGIA, CONGENITAL OCULOFACIAL PALSY, MÖBIUS SYNDROME

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Duke-Elder[20] states that paralysis of the seventh nerve in association with paralysis of one of the ocular motor nerves forms an interesting group of cases of central origin of which there are several examples. These include paralysis of the fifth, sixth, and seventh, or paresis of the third, seventh, and 12th nerves, but the most interesting lesion is a combination of the sixth and seventh, which occurs bilaterally.

This rare but interesting syndrome involving a failure of lateral movements of both eyes, associated with a bilateral facial paresis, is frequently known as the Möbius syndrome. It was noted and described in detail by Graefe,[21] Harlan,[1] Chisholm,[16] Möbius,[18] and others. In a typical case there is a complete bilateral facial paralysis with retention of Bell's phenomenon on attempted closure of the eyes. Lateral movements of the eyes both in adduction and abduction are impossible, so that the patient has to look to either side by turning his head. Occasionally uniocular adduction is possible, and a limited amount of convergence can be obtained, which is accompanied by contraction of the pupil. The patient can elevate and depress his eyes normally, but no vestibular nystagmus can be elicited by the calorie or rotary test.

Duke-Elder goes on to say that there is thus a failure in lateral movements of the eyes and in their vestibular connections, but there is retention of convergence and pupillary reactions. The muscles, nerves, and nuclei associated with the third nerve must therefore remain intact, and a destruction of the sixth and seventh nerves with an interruption of the vestibular pathway in the neighborhood of the posterior longitudinal bundle must be postulated. Whether this is due to an aplasia in this region, or a hypoplasia, or a localized fetal lesion is debatable.

Fig. 1 (Bedrossian and Lachman). Normal upward gaze.

Other associated congenital anomalies have been recorded, such as deformities of the ears, muscular defects of the tongue, neck, and chest, supernumerary or webbed digits, or absence of the fingers, and more particularly bilateral clubbed foot.

Henderson,[15] in 1939, reported 61 cases of the Möbius syndrome, in which 45 had abducens palsy. Of these 45 cases, 23 showed a convergent squint. The other 22 had straight eyes. In reviewing the literature concerning

Fig. 2 (Bedrossian and Lachman). Normal downward gaze.

Fig. 3 (Bedrossian and Lachman). Attempted right gaze. Note inability to abduct or adduct either eye.

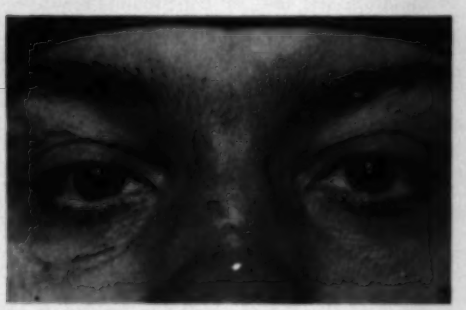

Fig. 4 (Bedrossian and Lachman). Attempted left gaze. Note inability to abduct or adduct either eye.

Fig. 5 (Bedrossian and Lachman). Attempted convergence. Note the constricted pupils and the slight inward movement of both eyes.

this syndrome, one of the most interesting features is the pathology that is postulated for these cases. The pathology seems to be explained by two schools of thought: One, as due to the existence of a primary aplasia of the muscle due to a mesodermic defect, and, two, as caused by a nuclear or ectodermal defect.

Evidence of muscle defects has been shown by Obersteiner,[23] Bernhardt,[24] and Heuck.[25] The most outstanding of these is the report by Heuck, in 1897, of a family in which a mother and three children all had eye muscle defects, including bilateral ptosis, inability for outward movement of either eye, as well as inability to elevate or lower the eyes. One of the children died at the age of 18 years, and the autopsy specimen showed that all rectus muscles except the medial rectus muscles were inserted abnormally posteriorly, some as much as 2.5 mm., and that nearly all of the muscles were too short, as much as 10 to 11 mm. in the case of the inferior rectus muscles. He concluded that the faulty insertions and incomplete development adequately explained the defective motility. The nerves were not examined.

Two main types of nuclear involvement are suggested by different authors. One type is that of aplasia of the nuclei. Huebner's[7] case report in 1900, which is referred to by Duke-Elder[20] and in Walsh's textbook,[22] is an example of this type. The other type of nuclear involvement is that of degeneration, as shown in a case reported by Rainy and Fowler[8] in 1903. In this case the patient had facial diplegia but no ocular involvement, and the pathologic study showed degenerated nuclei, nerves, and atrophied muscles. Other authors, such as Spatz and Ullrich,[26] and Bálint[27] have also reported nuclear pathology.

The possibility that two different types of pathologic processes may exist is nicely summarized in a very interesting article by Leszynsky[5] in 1897. He stated that, in the few cases examined post mortem, complete absence of the muscles or abnormal insertions were found. "Neuropathology teaches that fibrous degeneration and complete absence of a muscle may be due to a primary nuclear atrophy, while, on the other hand, the nuclear atrophy may result from a loss of function or atrophy of muscle."

Case report

The following case is an example of muscular pathology.

Mr. N. D., aged 35 years, complained of inability to move his eyes to the right or left since birth. Previous medical history was that of a left herniorrhaphy and spinal arthrodesis at the age of 16 and 18 years. Visual acuity was correctible to 6/6 in each eye. Ocular movements showed ability to elevate and depress the eyes normally. He was unable to abduct or adduct either eye. Convergence was partially present with normal pupillary reactions. His near-point of convergence was 20 cm. from the nose. Bell's phenomenon was present. The patient showed first, second, and third degree fusion in eyes front. His prism divergence was 10/5 and convergence 20/10. When tested for opticokinetic nystagmus on the rotating drum, there was no response on either side. His general findings were that of a slightly masked facies, suggesting facial paresis. He was able to smile slightly but not as most individuals. He showed diminished hearing on the right, a hammer toe on the right, and, in his family history, a cousin had webbed fingers.

A Bárány test was performed at another institution, and the report suggested a midline infratentorial lesion. In view of this finding, Bárány and caloric tests were repeated at the Graduate Hospital. The report of the consultant was:

"Hypoactive reflexes to both mass caloric and Bárány testing. Bárány testing is of decreased value because of lack of nystagmus. However, if the patient's eye complaints are on the basis of intracranial pathology, such pathology would be located in the midline to coincide with decreased response to vestibular stimulation."

Forced duction tests were performed under general anesthesia, and it was found that neither eye could be abducted or adducted beyond the midline with a forceps but we were easily able to elevate and depress either eye. The medial and lateral rectus muscles were then explored and found to have many check ligaments. Tenon's capsule in the region of the muscle insertions was about as thick as the muscle itself. The lateral rectus muscle was inserted 12 to 13 mm. from the limbus. The medial rectus muscles were also inserted slightly more posteriorly than normal. An attempt was made to stimulate electrically the muscles directly while under anesthesia. Both alternating and direct currents were used in the usual manner but no response was obtained. The technician performing the electrical stimulation felt that no conclusions could be drawn because the patient was under anesthesia.

Following surgery, in which both medial and lateral rectus muscles were explored in both eyes, and all adhesions and check ligaments were freed, the patient was still unable to abduct or adduct either eye. There was, however, some improvement in his near-point of convergence.

We feel that this case is of great interest because of the definite inability to abduct or adduct either eye under forced duction tests, and also because the muscles themselves were attached posterior to the equator and, therefore, had no mechanical advantage for rotation. This would suggest that in our case, the pathologic process was peripheral in the muscles and their attachments and probably not in the nuclei of the brainstem.

Comment

1. Bilateral paralysis of abduction and adduction may occur as an acquired condition from central nervous-system disease. The clinical findings in such a case may resemble those of congenital paralysis of abduction and adduction.

2. The pathology in congenital cases is thought to be due to nuclear aplasia (or degeneration) or muscular aplasia. The forced duction test should be very useful in differentiating peripheral muscular defects from a nuclear lesion in the congenital cases.

3. The case presented here shows the

pathologic process to be definitely peripheral in the muscles. It is hoped that in the future more cases of this type will be recognized and that forced duction tests and exploration of the muscles will be performed for diagnosis. If this is done, then future correlation of muscular aplasia with nuclear aplasia may be obtained.

4. A knowledge of existent peripheral congenital pathologic conditions of the ocular muscles will help prevent misinterpretation of vestibular tests in suspected cases of brain tumor.

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UNILATERAL RETINITIS PIGMENTOSA*

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Retinitis pigmentosa is a chronic progressive noninflammatory disease of unknown origin. It may be discovered in early childhood and ultimately results in blindness after middle age. There is usually a hereditary history. The disease is usually bilateral and both eyes are equally affected.

Pathologically it is a degeneration of the neuroepithelium, especially of the rods, and is accompanied by attenuation of the retinal vessels and finally results in optic nerve atrophy.

Pigment changes of the retina were known even before the days of the ophthalmoscope (Schoen, 1828). In 1861, Liebreich found pigmentary degenerative changes of the retina in certain families especially among first cousins. Nettleship (1907-1908) found 976 families with this disease and hereditary transmission was found in half the cases. There was no history of consanguinity in 23.5 percent, with consanguinity 23 percent, and 3.5 percent with both heredity and consanguinity.

It may appear as a dominant, a recessive, or as a sex-linked trait. The first trait which is rare may appear in larger and more heavily affected families; the second is most common; the third is very rare.

Both sexes transmit and are equally affected with the disease as a dominant characteristic. In the second instance, parents transmit recessive traits jointly. Some believe consanguinity plays a very important role. The sexes are equally affected. As a sex-linked trait, the disease is transmitted by females and appears in males only.

When retinitis pigmentosa is a dominant characteristic the affected individual gets this disease directly from one parent and it usually passes on to half of the children. When it appears as a recessive trait it is imperative that intermarriage be avoided within the same pedigree, or into a family carrying the trait. If such marriage does occur, sterilization should be done (Duke-Elder). When retinitis pigmentosa is inherited as a recessive sex-linked character, half of the sisters and all the daughters are affected. Males are more frequently affected. The responsibility therefore rests upon the women of the family.

Associated degenerative conditions may include myopia, deafness, mutism, mental inferiority or insanity, multiple sclerosis, and constitutional and skeletal evidence of pituitary dysfuncton.

Predisposing causes are unknown but precipitation or acceleration of symptoms are usually associated with severe illness or massive loss of blood. Acute xanthema (Nettleship, 1907-1908), malaria (Smith, 1904) Vincent's angina (Henderson, 1934), syphilis or tuberculosis apparently has no association with retinitis pigmentosa.

Unilateral retinitis pigmentosa is extremely rare. Pedraglia, in 1865, and de Wecker, 1868, were the earliest to report unilateral retinitis pigmentosa. Shoji, in 1926, followed a case of unilateral retinitis pigmentosa for 10 years.

In some instances clinical evidence of this disease has been found between three and eight years of age (Horing, 1864), and usually serious symptoms begin during early school age, between the age of six and 12 years. By 20 years of age, the patients show

* From the Department of Ophthalmology, The New York Medical College.

signs of incapacitation. The age of blindness varies, it usually occurs between the age of 40 and 50 years, rarely beyond 60 years.

Attenuation of the retinal vessels and pigmentary changes are constantly present. The arteries are especially affected and may even appear thready. The disc becomes waxy and atrophic. The pigment becomes aggregated into characteristic jet-black specks, spidery in shape, resembling bone corpuscles, which cluster around the retinal veins and migrate into the retina itself. As the migration of pigment progresses, the pigment epithelium becomes more decolorized and the fundus appears tessellated; the choroidal vessels come into view more clearly, first at the equatorial region.

The extreme periphery and the central area of the fundus remain quite normal but, as the disease progressess, the entire retina becomes thickly studded with pigment masses, impairing vision. Other changes are high myopia and posterior polar cataract due to malnutrition of the lens. Glaucoma may sometimes occur.

Symptoms. Night blindness is the earliest complaint in youth and may be present for several years before pigment changes are observed.

There is usually an annular or ring-shaped scotoma corresponding to the degenerated zone of the retina and, as the disease progresses, it widens out, anteriorly and posteriorly. The corresponding field disappears, quadrant by quadrant, the lower temporal periphery remaining the longest until a small area around the fixation spot is left. Tubular vision is present and eventually central vision is lost, so that even light perception is lost.

The clinical course is slow, chronic, and progressive and may be interrupted by remissions in which the visual acuity and fields show marked improvement. These remissions may frequently be attributed to some varied forms of treatment which the victim receives.

Atypical pigment degeneration. In central pigment degeneration there are islands or clumps of black specks around the macula and central vision is lost early.

Degeneratio sine pigmento. The clinical picture and symptomatology of pigment degeneration may appear without any or a very minimal degree of fundus changes.

Acute pigment degeneration. Described by Stock in 1936 were three children of the same parents whose disease started in the sixth year of life and terminated in total blindness. However, the entire retina was affected simultaneously which differs from a true retinitis pigmentosa. Dementia and epilepsy due to cerebral degeneration were also present.

Pathologic examination in primary pigment degeneration of the retina reveals a progressive degeneration of the neuroepithelium, primarily of the rods, followed by general atrophy of the whole retina associated with an overgrowth of glial tissue or depigmentation of the retinal epithelium with migration of pigment into the retina, with an obliterative sclerosis of the retinal vessels.

Etiology. It is believed by most ophthalmologists that primary degeneration of the neuroepithelium and pigmentary epithelium is the etiologic factor. Some believe that sclerosis of the choroidal vessel may have an etiologic role. Disturbances of the sex hormones accentuate the disease at puberty. Endocrine dyscrasias, pituitary disturbances, hepatic disease, vitamin deficiency, and toxic factors may also have etiologic significance.

Treatment has been disappointing. Vasodilators, retrobulbar injections of atropine, miotics, trephination, cyclodialysis, section of the sympathetic, hormones, liver extract, galvanism, implantation of fresh placenta into the rectus abdomonis are only a few of a great many remedies which have been tried. Unfortunately all these treatments have had no effect. At present no magic formula has been devised to aid those suffering from this disease. The natural fluctuation of this disease and the hopefulness of the patient, as well as the enthusiasm of the ophthalmologist, have fostered a number of optimistic "preliminary" reports.

Case report

Mrs. J. B., aged 28 years, married five years, was a healthy appearing woman. She had never been pregnant. When she consulted me in October, 1948, she gave a history of poor vision at dusk, for three years.

Examination revealed visual acuity: R.E., 20/30; L.E., 20/25. There was a low error of hyperopic astigmatism in each eye. The lids were negative, muscles showed no deviation, cornea and lenses were clear. Discs were ovoid and, in the right eye, off color temporally. The vessels appeared slightly attenuated. In the right eye, toward the periphery there were black, jetlike pigment deposits surrounding the larger vessels. Peripheral fields were concentrically contracted to about 50 degrees. Left eye: cornea and lens were clear. Fundus examination showed: disc was ovoid, of good color, physiologic cupping, vessels were of good caliber. There was no evidence of any pigmentary changes throughout the retina.

A diagnosis of retinitis pigmentosa was made.

In 1951, a re-examination was made. The appearance of the right fundus was practically unchanged. There was no evidence of any pigmentary degeneration in the left fundus. The caliber of the vessels was well within normal limits. There was no gross pathologic process. In the right fundus toward the periphery there were the usual black jetlike pigment deposits surrounding the larger vessels. The peripheral field was contracted concentrically to about 45 degrees.

In 1953, at re-examination, the appearance of the right fundus was practically unchanged. The left fundus showed no evidence of any pigmentary change. After five years, there was no evidence whatsoever of any pigmentary degenerative changes in the opposite eye.

525 Park Avenue.

POSTNATAL CATARACTS IN A PREMATURE INFANT

Hugh Ryan, F.R.C.S.
Melbourne, Australia

With the increased interest in the eyes of premature infants that has followed the investigation of retrolental fibroplasia, several interesting conditions, some pathologic and some now known to be normal, have been described. Among the former is the discovery that cataracts can develop in early postnatal life in premature infants without any apparent cause. Two cases were reported by Guy (1954) in which he was able to exclude the more usual etiologic factors of trauma, uveitis, galactose disease, malnutrition, endocrine disturbance, and radiation. In a footnote he mentioned a third case. An additional case is here recorded.

Case history

R. H., a boy, was born on May 25, 1952, after 36 weeks' gestation and weighed five lb. (2,268 gm.). The mother was aged 31 years, had two live children and had had six abortions previously. These had occurred during the third and fourth months of pregnancy. She was Rh positive and had a negative Kline test. She was admitted to the Royal Women's Hospital, Melbourne, with the diagnosis of pre-eclamptic toxemia in the eighth month of her ninth pregnancy. The membranes were ruptured and a normal delivery followed.

The baby was rather edematous and anemic at birth, the bridge of the nose was depressed, there was a systolic cardiac bruit, and there were large indirect inguinal hernias. It was artificially fed and in addition was given ascorbic acid, 25 mg. twice daily, tablets of mixed vitamin-B group, vitamin B_{12}, folic acid, and thyroid (gr. 1/60, [1.0 mg.] daily).

On June 6, 1952, the serum protein was 4.40 percent and microscopic examination of the urine showed a few pus cells and some organisms identified as Staphylococcus au-

reus. On June 9th, ecchymoses were noticed about the eyes and nose. The fundi were examined under homatropine and cocaine mydriasis on June 13th, and showed grayish elevations at the periphery, as is frequently seen in premature infants, but the lenses were clear. Next day the infant was found to have a temperature of 102°F. (38.9°C.), but this subsided the following day.

On June 17th, the infant's hemoglobin was found to be 8.1 gm. percent (56 percent), and it was given a transfusion. On June 23rd, it again had an elevation of temperature of 101°F. (38.3°C.), and on June 26th, the baby had diarrhea. It was given methionene (125 mg. a day), penicillin, streptomycin (30 mg., twice a day), phenobarbitone (0.5 gr. [0.01 gm]), gardinal (3.0 gr. [0.19 gm.]), and chloramphenicol (25 mg. eight hourly).

On June 27, 1952, the temperature was 104°F. (40.0°C.), and examination of the stools showed no pathogenic organisms but, on June 30th, blood was seen in the stools. The condition slowly subsided but, on July 7th, a temperature of 100°F. (37.8°C.) was found and examination of the chest showed rhonchi. On July 17th, the hemoglobin had fallen to 7.6 gm. percent and a transfusion of eight oz. (227 ml.) of blood was given. By July 31st, hemoglobin had again fallen to 7.5 gm. percent and another transfusion of four oz. (114 ml.) was given.

On August 18, 1952, the eyes were examined under full mydriasis and bilateral mature cataracts were found. A hemoglobin estimation gave a reading of 8.2 gm. percent on this date and a further transfusion of six oz. (170 ml.) was given. By September 12, 1952, there was again an attack of diarrhea and, on September 18th, a transfusion of five oz. (142 ml.) of blood was given because the hemoglobin had again fallen and was now 8.1 gm. percent. Urine examination on this date showed some pus cells and a few B. coli but no reducing agent for Benedict's solution was found.

The baby was discharged to home on September 25, 1952, where it continued to progress in a satisfactory manner. During its stay in hospital it had received only a little oxygen. Bilateral discission of both lenses was performed under ether anesthesia on March 10, 1953, but there was little absorption of the lens and it was repeated on July 21, 1953. In the early hours of the following morning the infant became convulsed and cyanotic and had a temperature of 104°F. (40°C.) and a pulse rate of 150 per minute. It remained in this state for 24 hours and died on July 23, 1953.

Post-mortem examination showed consolidation of the base of the right lung with patchy areas of consolidation over both lungs. There was a mucopurulent exudate in the small bronchi. The liver showed fatty degeneration and the abdominal lymph nodes were enlarged. The spleen weighed 30 gm. and showed follicular hyperplasia. The thymus weighed 25 gm. The pituitary, pancreatic, thyroid, and adrenal glands were normal. The kidneys were normal. The brain showed some congestion but was otherwise normal. The eyes could not be obtained for histologic examination.

The cause of death was pneumonia and pulmonary collapse. No evidence of metabolic disorders such as galactosemia could be found to account for the cataracts.

Discussion

The observation of cataracts developing in early infant life is very rare. However, it may be more common than is thought, for it is only by routine examination of all premature babes at birth, or soon after, that it is possible to prove that the lenses were clear at birth and that any cataracts observed later developed in postnatal life. At the Royal Women's Hospital, Melbourne, the eyes of all premature babes whose birthweight is under 3.5 lb. (1,600 gm.) are examined routinely under homatropine and cocaine as soon after birth as possible, and at intervals until discharged from the premature babies' ward. They are later examined at a special

ophthalmic follow-up clinic. Since this procedure was instituted in September, 1949, the number of premature infants under 3.5 lb. (1,600 gm.) examined, and who have survived to leave hospital is 430. Only one case of postnatal cataract has been observed in these 430 infants. The incidence is therefore very rare.

The outstanding features of this case are the appearance between the sixth and tenth week after birth of bilateral cataracts and their rapid maturation (in less than four weeks) in a premature infant suffering from gastroenteritis and anemia, but otherwise a healthy child. The etiology of this type of cataract is most probably toxic in nature, but the exact nature of the toxic responsible will be undetermined until more cases are reported.

Duke-Elder (1940) describes such bilateral cataracts as occurring in acute toxic illnesses. He states that usually they occur in the cortex and mature rapidly, but in most cases the illness is of a very severe nature.

Guy (1954) considered that oxygen therapy might be a factor as in retrolental fibroplasia. However, this is unlikely, since cataract is conspicuous by its absence in retrolental fibroplasia and the deleterious effect of excess oxygen therapy is felt most by the retinal vasculature. Again, this infant received very little oxygen. Prematurity of itself would be unlikely, because of the rarity of the condition in contrast to the frequency of prematurity, but it is certainly a predisposing factor.

Mann (1937) describes two forms of postnatal developmental cataracts, one associated with pathologic states due to endocrine imbalance and the other showing no such association. These cataracts, however, are more or less confined to the lens fibers which are produced after birth, but in the case here presented all the lens fibers were involved and the appearance does not resemble those described by Mann. Dehydration as a cause must be considered but such dehydration as existed was unlikely to have produced the damage.

The infant received much therapy, including ascorbic acid, mixed vitamin-B tablets, vitamin B_{12}, folic acid, thyroid, methionine, penicillin, streptomycin, phenobarbitone, gardinal, chloramphenicol, and transfusions, but this is a common experience for many premature infants who show no ill effects from it.

Again mild attacks of gastroenteritis are not uncommon among premature infants and anemia is present quite frequently. In the first case reported by Guy (1954), both these symptoms were present.

Summary

The cause of these cataracts is probably a toxin, in this case arising from the bowel, acting on an individual weakened by prematurity and anemia. No other cause could be found during life or at autopsy but, before the cause can be definitely established, further cases must be reported and the group analyzed.

5 Collins Street.

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A BURIED TYPE OF INTEGRATED ORBITAL IMPLANT*

RICHARD BUTTON, M.D.
Newport Beach, California

INTRODUCTION

The great volume of literature written on the various types of orbital implants lends support to the fact that as yet there has been no completely satisfactory implant which can remain covered and yet duplicate exactly the movements of the normal human eye. The ideal implant should be covered with conjunctiva, have mobility, and be securely imprisoned by the ocular muscles. It should also be completely integrated with the overlying prosthesis.

The idea of an integrated orbital implant was originally introduced by Ruedemann[1] in 1945. His implant was a combined eye implant which was attached directly to the extraocular muscles. The principal disadvantage was infection and subsequent extrusion of the implant, since it was not covered by conjunctiva. Subsequent modifications of this type of integrated orbital implant came from Cutler,[2] Stone,[3] Albaugh,[4] and many others. The Troutman magnetic implant[5] was a further modification with the important advantage of being completely covered with conjunctiva, thus decreasing the danger of infection and extrusion.

The continued use of integrated orbital implants has been encouraged by the fact that muscles can be permanently attached to inert foreign material, such as metallic mesh and plastic. However, the attachment of living tissue to a foreign material leaves much to be desired and greatly enhances the danger of extrusion of the implant. The implant described in this report utilizes the sound surgical principle of suturing tissue to tissue rather than to an inert foreign material.

SURGICAL PROCEDURE

The implants were made of the inert acrylic resin, methyl methacrylate, and were duplicated from an original experimental wax model. The implants were constructed with a detachable top incorporating a three-mm. nipple to fit the under surface of the prosthesis. This top was threaded and could easily be screwed into the matching base of the implant as shown in Figure 2. The base of the implant was constructed with four tunnels through which the ocular muscles could be introduced and secured in place.

The patients were prepared in the usual manner and the anesthesia was left to the discretion of the surgeon. A lid speculum was used to separate the lids as shown in Figure 1 and a circumcorneal conjunctival incision was made as close as possible to the limbus to preserve as much conjunctiva as possible. This incision was carried posteriorly exposing all of the rectus muscles which were in turn isolated, and a double-armed, 4-0 chromic suture was threaded through the tendinous insertion of each rectus muscle and locked at both ends, as shown. Each of the muscles was then severed from its attachment and the enucleation was continued in a routine fashion.

After hemostasis was obtained the proper-sized orbital implant was selected as shown in Figure 2. In adults, a 17.5-mm. implant was used and in children a 14-mm. implant was recommended.

The base of this implant was then introduced into the orbital defect as shown in the drawing and the rectus muscles were individually threaded through the tunnels in the base of the implant. The muscles were sutured in pairs, lateral rectus to medial rectus and superior rectus to inferior rectus. This is well illustrated in Figure 3, showing the rectus muscle being secured in place

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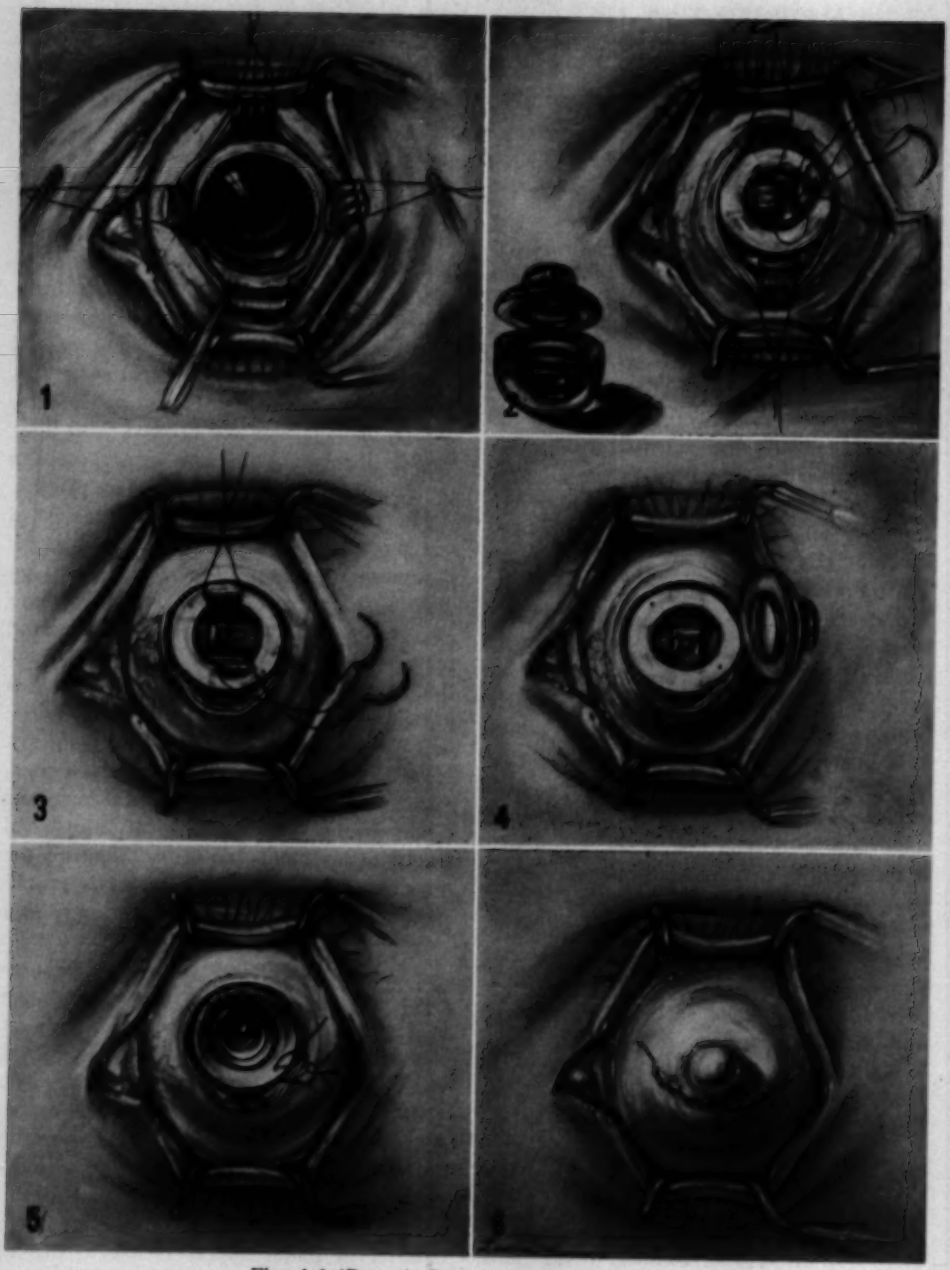
* From the Department of Ophthalmology, Fort Defiance General Hospital, Fort Defiance, Arizona. I would like to express my gratitude to Dr. Nicholas Zrinyi and Dr. Norman Buckman of the Dental Department, for their co-operation and assistance in preparing the experimental implants and prosthesis. Acknowledgement is also made to Mr. Zolton Yuhasz for his very excellent illustrations.



Figs. 1-6 (Button). Steps in operative procedure.

within the base of the implant. In Figure 4 the four rectus muscles can be seen sutured in place. In Figure 5 the detachable top of the implant is being screwed in place with the aid of an assistant who immobilizes the base. The top is then fixed in place with a thread of stainless steel wire, which locks the top and base together.

Figure 6 represents the final and most important stage of the procedure. In this stage the operative wound is closed, and it is imperative that all of Tenon's capsule be brought together and sutured over the implant. This is sutured separately with interrupted 4-0 chromic suture and the conjunctiva is closed over this. If there is too much tension on the closure then the implant is too large and should be replaced with the correct sized implant or the detachable top should be removed converting the procedure into a conventional type of basket implant which can easily be covered with conjunctiva. If the correct implant has been selected then the conjunctiva may easily be closed, without tension over the protruding nipple, with a running silk suture as shown.

Aureomycin ophthalmic ointment is instilled into the conjunctival sac and a semi-pressure dressing applied to the eye. A conformer is not recommended in these cases.

Discussion

The value of the implant described in this report lies chiefly in the fact that it is completely buried and offers good motility to the overlying prosthesis. If the nipple erodes through the covering of conjunctiva then the detachable top may be removed quite simply without disturbing the base of the implant which can be covered easily with conjunctiva.

The patient is not fitted with a prosthesis until the operative wound has thoroughly healed. Usually by the end of the third postoperative week the patient can be fitted and at this time an impression can be taken of the socket with dental compound. From this compound a rough prosthesis can be fashioned which is used as a final model from which the actual prosthesis is made. The prosthesis must exactly fit the protruding nipple of the implant or erosion will occur.

Complications

Most of the complications experienced with this implant directly concerned the erosion of the nipple through the overlying conjunctiva. This was primarily due to the choice of too large an implant thus producing tension on the suture line. Careful selection of an implant will minimize this complication especially in children. In no case has there been either infection or extrusion of the implant after a period of 12 months. In one of our cases it was necessary to remove the detachable top since it had eroded through the conjunctiva but the final result has been satisfactory since the buried base of the implant has remained secure and motile.

Summary and Conclusions

A total of six cases have been performed utilizing this implant with erosion occurring only in one child in which too large an implant had been used. The results with this implant have been very encouraging although the number of cases are too few to be conclusive. The only complication experienced to date has been erosion of the tip through the conjunctiva. No final evaluation can be made until more cases have been collected and have passed the five-year evaluation period.

419 North Newport Boulevard.

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A CORNEOSCLERAL EPITHELIAL INCLUSION CYST*

A RARELY DESCRIBED CLINICOPATHOLOGIC ENTITY

ALBERT D. RUEDEMANN, JR.
Detroit, Michigan

CLINICAL

G. H., a 23-year-old white man, was seen because of a mass on the left eye which has been present since birth. At first it was only a "water blister." No treatment was attempted until he reached the age of three to four years when a local doctor prescribed "drops." These were useless and treatment was discontinued. The mass gradually enlarged to the present time and the patient was disturbed by the cosmetic appearance as well as by some irritation (fig. 1).

Eye examination. Vision was: R.E., 20/20, corrected; L.E., 20/200, no improvement with glasses or pinhole.

External. R.E.: Essentially negative. L.E.: Negative except for the mass as seen in Figure 1. Pressure with a glass rod could transmit intracyst fluid from corneal to scleral portion.

Tension was (Schiøtz): R.E., 20 mm. Hg; L.E., 20 mm. Hg.

Slitlamp examination was essentially negative. L.E.: Cornea clear except for a clear walled cyst extending nearly to the center of the pupillary space on the corneal side and approximately 4.5 mm. on the scleral side. Fluid in the cyst was clear and there was a definite fluid level of debris in the corneal section of the cyst. This was a chalky white color and may have been cellular debris. Remainder of anterior ocular segment negative.

Ophthalmoscopic examination (dilated) was essentially negative, O.U.

Operative note. Using local anesthesia, the conjunctiva and episcleral tissue were incised by a horizontal incision and the anterior layer of the scleral cyst was removed intact using scissors and forceps. The posterior layer was left in place for fear of further weakening of scleral wall. Then an attempt was made to scrape the epithelial lining of the posterior scleral layer and the corneal section of the cyst, using a blunt dissector. (Biochemical evaluation of the cyst fluid was not available.) The connecting portion of the cyst at the limbus was then firmly sewed to the deeper layers using interrupted 6-0 sutures. One drop of one-percent atropine was instilled, followed by antibiotic ointment. An eyepad was applied.

Postoperative course. Recovery was uneventful. The corneal cyst gradually re-

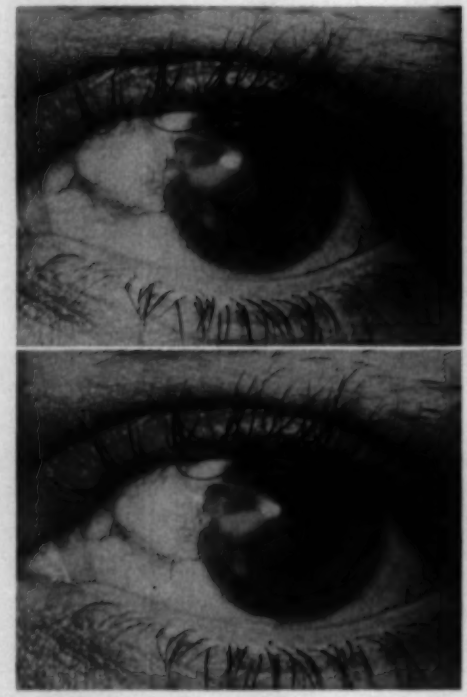

Fig. 1 (Ruedemann). Place five prism diopters base-up over one eye and five prism diopters base-down over the opposite eye, or hold sideways and stereo. Note white chalky epithelial debris within cyst. Limbal connection between cornea and sclera is at the 9:30-o'clock position. (Donaldson camera.)

* From the School of Aviation Medicine, Randolph Air Force Base, Randolph Field, Texas.

Fig. 2 (Ruedemann). High power. Hematoxylin and eosin. Section of subconjunctival cyst, showing fibrous scleral wall and epithelial lining.

turned but only to about one-half the original size. A small hole appeared about two mm. from the limbus over the scleral portion. It did not appear to be connected to the corneal portion because fluorescein gave no evidence of flow and direct pressure to the corneal cyst could not elicit fluid. Vision improved to 20/60 and the patient stated that there was very little irritation. He has not been seen again.

PATHOLOGY (fig. 2)

Diagnosis. Subconjunctival tissue, left eye: Cyst, congenital, sclera and cornea. Examined by: James R. Duke, 1st Lieut. (MC) U.S.A., Fitzsimons General Hospital, Denver, Colorado.

Comment. This cyst might simply be called a scleral cyst as it resembles this type of cyst as described briefly in the *Atlas and Textbook of Ophthalmic Pathology.* Also, it could have arisen from a misplaced rest of conjunctival epithelium. In this instance, it would be analogous to the epidermal inclusion cyst which is often found located deep in the dermis.

Gross. This specimen fixed in formalin consists of a small flat piece of tissue, roughly square, measuring 4.0 by 4.0 mm. Two opposing edges are folded slightly suggesting that this was originally a cyst wall. The tissue is gray and translucent and several vascular channels may be seen in its wall. The entire specimen is submitted for embedding.

Microscopic. The tissue is composed of a moderately thick wall of fibrous tissue and collagen fibers. Very little elastic tissue is noted in this wall. Occasional small vascular channels are noted. One surface of this wall is lined by a layer of epithelial cells, three or four cells in thickness. The superficial cells tend to be flattened or squamous, whereas the deeper cells and more basal cells are round, oval, or polygonal in shape. No glandular structures or hair follicles are noted.

David Whitney Building (26).

Dr. Paul Cibis, School of Aviation Medicine, Randolph Field, Texas, suggested the use of vertical prisms to superimpose the stereopictures.

OPHTHALMIC MINIATURE

July 5 (Lord's day). About four in the morning took four pills of Dr. Turberville's prescribing, for my eyes, and they wrought pretty well most of the morning.

Pepys' Diary, 1668.

OPHTHALMIC RESEARCH

Department

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers read at the VIII Annual Clinical Conference of the Staff and Wills Eye Hospital Ex-Resident Society

Philadelphia, Pennsylvania
February 17 and 18, 1956

The effect of Chlorpromazine upon the intraocular pressure of experimental animals. Satya Dev Paul, M.D., and Irving H. Leopold, M.D.

Thorazine (Chlorpromazine) was administered by intramuscular injection and topical application to the eye. Various dosages from 1.0 mg. to 100 mg. were used in 66 rabbits and 18 cats.

In the lowest dosage the fall in intraocular pressure was within experimental error. Doses of 50 to 100 mg. produced significant fall in intraocular pressure in both rabbits and cats. The effect of the drug on the pupil in these two groups of animals was varied. In rabbits it produced miosis and in cats mydriasis.

Local application of the drug had no hypotensive effect and was irritative to the eye. The site of action of this hypotensive agent was not determined.

The effect of age upon lens metabolism. Harry Green, Ph.D., Shirley A. Solomon, A.B., and Irving H. Leopold, M.D.

The metabolic activity of lens obtained from three-year-old rabbits was compared with that from three-week to one-month-old rabbits, with respect to the ability to convert glucose to lactic acid under anaerobic conditions. The results with cell-free lens extracts show that the old lens can metabolize fructose diphosphate as readily as the young lens, but that there is a diminished ability to convert glucose to fructose diphosphate. Thus it appears that, in the aging of a rabbit lens, there is an associated slowing down of the anaerobic carbohydrate metabolism.

C-reactive protein as applied to clinical and experimental uveitis. Michael I. Wolkowicz, Joseph W. Hallett, M.D., and Irving H. Leopold, M.D.

Blood serums of 103 patients were tested for the presence of C-reactive protein. The results emphasize the prevalent opinion that in a large percentage of patients with uveitis an association between uveal tract inflammation and systemic disease can be demonstrated. Studies of the aqueous humor of rabbits' eyes suggest that the C-reactive protein determination may be helpful in classifying the miscellaneous group of uveitis on the basis of etiologic factors.

The influence of cyclodiathermy on choroidal circulation. Gerard M. Shannon, M.D., and Irving H. Leopold, M.D.

The effect of angiodiathermy on intraocular pressure was studied in experimental animals and evaluated by three techniques. The tension was measured by a Schiøtz tonometer and the untreated eye used as a control in each animal. The tension was recorded before application

of diathermy and at appropriate intervals thereafter. At each interval when the tension was recorded, using the scleral dehydration technique, Kodachrome transparencies were made, thus recording the changes in choroidal circulation. The treated eyes were then injected with a plastic preparation and the specimens corroded, allowing the cast to be evaluated for changes in the choroidal circulation. Lastly, a series of specimens was prepared and stained for histologic study.

The study indicates that the influence of diathermy upon the intraocular pressure depends on whether the arteries or veins are treated. When the veins are treated, there is a rise in intraocular pressure which may be due to edema and engorgement. This soon subsides and the tension returns to levels similar to those found preoperatively. When the arteries are treated, a transient initial rise in intraocular pressure occurs. It is less than is seen when veins are treated. In a few animals a fall in tension occurred for short periods of time. When the anterior segment is deprived of its blood supply, necrosis results. It was also observed that, although the blood flow is re-established in three to four weeks, the ciliary body involved shows the effects of the angiodiathermy for several months.

Observations on scleral resection. Enrique Wudka, M.D., and Irving H. Leopold, M.D.

Lamellar scleral resections were performed in albino rabbits and their progress followed by microscopic and ophthalmoscopic observations. Unfolding of the resected sclera occurred in one to two weeks after surgery. Local and general changes of a transitory nature were observed in the choroidal circulation. Temporary detachment of the retina occurred in the resected area.

The beneficial effects of the operation are believed to be due to transitory approximation of the external ocular coats to the detached retina.

Treatment of iris cysts with electrolysis. Patrick J. Kennedy, M.D.

Six cases in which treatment of iris cysts by electrolysis was successful are reported. Four of these occurred after cataract extraction. One case occurred spontaneously in an 18-year-old female. Two of the cysts required two treatments. The only complication was a mild iritis which responded rapidly to therapy.

It is believed that electrolysis offers the best and safest method at the present time for the treatment of iris cysts.

A plastic operation for enucleation of the eye. Edmund B. Spaeth, M.D.

The procedure consists of simple enucleation and insertion of an implant into the socket and securing a small mucous membrane graft to the under surface of the conjunctiva.

This mucous membrane graft when attached to the conjunctiva forms a small pocket which receives a short curved hook which is fastened on the back of a temporary conformer and later on the back of the prosthesis. Inserting the hook into this small pocket provides better rotation of the prosthesis.

Beta irradiation as an adjunct to glaucoma surgery in the Negro. Wilfred E. Fry, M.D., and Louis B. Cohen, M.D.

It is generally recognized that the results after glaucoma surgery in the Negro race are not as satisfactory as in other races. The deeper pigmentation found in the iris may be a particularly important factor.

After performing the iridencleisis operation in these patients, beta irradiation is immediately applied to the wound. This provides better results than were obtained otherwise.

An initial evaluation of prednisone therapy. James F. O'Rourke, M.D.

Prednisone has five times the potency of cortisone and hydrocortisone in both the prophylactic and the active treatment of acute animal uveitis. The time required for clearing and the onset of therapeutic effects is not apparently decreased.

Clinical studies indicate that the best results are obtained in acute nongranulomatous iridocyclitis. The over-all response of chronic lesions of the anterior segment is not encouraging.

A selective response to one steroid only was exhibited by certain posterior-segment lesions. This is interpreted as an expression of lesion or species specificity. Such specificity might explain the failure of prednisone to elicit a response in certain cases. There is a paucity of side effects.

Eye disorders associated with neurosurgical diseases of the brain and nervous system. Rudolph Jaeger, M.D.

Eye disorders of various kinds are cared for by the neurosurgeon. They are to be differentiated from those conditions which are purely ophthalmic. There can be no sharp line of division where one specialist leaves off and another takes over. Certain conditions require surgical therapy even after the ophthalmologist has made a precise diagnosis.

The major problems are associated with tic douloureux, intracranial aneurysm, cerebellar pontine tumor, and cancer around the orbit. Motor disturbances, such as extraocular palsy, caused by tumor in the orbit and cranial cavity must be cured by surgical methods. Surgical treatment for the various facial tics holds some promise for partial relief.

There are many surgical problems associated with lacrimation, the phenomenon of Horner's syndrome, and migraine. A much larger problem is that associated with a loss of vision by pressure on the optic nerves and visual centers and by choked discs.

Sodium saccharine for testing the patency of the lacrimal passages. Edward I. Lipsius, M.D.

The patency of the lacrimal passages was studied by instilling a 10-percent sodium-saccharine solution into the conjunctival sac. Sodium saccharine is used because of its easily detected sweetness, which is not unpleasant, and because of its solubility. After passing through the lacrimal passages and the nose into the throat, the solution may be tasted. It is soluble in a 50-percent solution which may be instilled into the eye but is somewhat irritating. The 10-percent solution is used because it is only mildly irritating and is still sweet enough to be identified.

The advantage of the test is that it simulates physiologic conditions, whereas forceful irrigation with a syringe and lacrimal needle does not. Successful irrigation may not, in some cases, prove there is adequate tear drainage. In three percent of cases in which there is no clinical disturbance of lacrimal drainage and no tearing, the patient is unable to taste the saccharine.

When the test is positive, the saccharine is usually tasted in five to 17 minutes but it may take nearly an hour. Usually the sensation is that of sweetness but some patients find the taste bitter and others are unable to identify the taste, although they note that they taste something different. The taste, usually described as coming from near the nose, may persist for many hours. Because of this, only one side can be tested by this method in one day.

A false positive test may occur if the patient gets the saccharine on the fingers and transfers it to the mouth. At present, this test is advocated only as an easily performed qualitative test which has no serious complications.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

MEMPHIS EYE, EAR, NOSE, AND THROAT SOCIETY

MALIGNANT EXOPHTHALMOS TREATED WITH ACTH AND CORTISONE

DR. H. CONDRON presented a 57-year-old white man who was admitted to the Kennedy Veterans Hospital on February 19, 1954, as a transfer from the Veterans Hospital in Marion, Illinois. The patient's complaint was marked swelling of both eyes, lids, and protrusion of both globes. His history dated back to May, 1953, when he first noticed the lids of both eyes swelling. This was gradually followed by developing exophthalmos in both eyes. At first the vision did not diminish; in fact, both eyes "tested 20/20 in January, 1954."

Because of the continued exophthalmos, the patient saw a private physician in July, 1953, who fitted him for glasses. This did not improve his condition so the physician placed him on eyedrops, the nature of which is not known. This likewise did not cause any improvement.

The patient was then hospitalized for a diagnostic work up. However, nothing positive was found. The exophthalmos and swelling continued and he was next sent to the McMillian Clinic in St. Louis. Here also nothing of diagnostic significance was found.

From the McMillian Clinic the patient entered the Marion Veterans Hospital in Marion, Illinois, in February, 1954. From this hospital he was transferred to Kennedy. He states that, since January, he has had marked visual loss in both eyes but has not noticed any great progression of the exophthalmos.

Physical examination. A well-developed, 57-year-old white man, slightly obese, in no acute distress. No gross abnormality of gait, status, or body symmetry. Grossly, there is evidence of bilateral exophthalmos and rather marked swelling of the lids of both eyes. Blood pressure is 140/80 mm.Hg. The heart sounds are regular in force, rate, and rhythm. No murmurs were audible. The lungs seemed to be clear. Abdominal examination revealed well-healed, bilateral herniorrhaphy scars; no organs were palpable.

Laboratory findings. Complete blood count: within normal limits. Serologic test for syphilis: negative. Urinalysis: negative. NPN: 27 mg. percent. Fasting blood sugar 72 mg. percent. The blood calcium was 5.1 mEq/l. The inorganic phosphorous was 1.65 mEq./l. Total protein 6.5 gm. percent. Albumin 5.9 and globulin 2.6 percent. The cholesterol was 205 mg. percent.

Eye examination. Special eye examination at the time of admission revealed the following: Vision: R.E., 18/400, pinhole no change, with glasses 20/400, JO; L.E., 15/400, pinhole 20/400, with glasses 20/400 and JO. Correction was: R.E. +3.5 D.sph.; L.E., +2.5 D.sph.; with a +1.75 add.

External examination. Palpebral openings were 17 right and left. Exophthalmometer reading: right 24, left 24 with a base of 108 mm. Extraocular muscle examination showed limitation of all movements, especially severe limitation of upward gaze. Tension was 25/37 mm. Hg (Schiøtz). The pupils were small but reacted slightly to light. It may be noted that the patient was on pilocarpine. There was swelling of the lids of both eyes and moderate chemosis and conjunctival injection. The globes were firm and did not compress easily into the orbit. Corneas of both eyes were clear and the lids closed normally over the corneas. No corneal exposure was evident. The anterior chambers were deep.

Internal examination of the eyes. The veins appeared dark and slightly engorged. No papilledema was evident. The entire fundus could not be visualized due to the small pupil. There was cupping of both nerves; however, it did not seem to be glaucomatous in type. Fields showed a relative scotoma, 15 degrees in diameter, centrocecal in position, in the left eye. In the right eye there was a 20-degree temporal contraction with an inferior quadrant contraction extending to the point of fixation. The blind-spots could not be plotted. The initial eye examination seemed to point to a thyrotropic type of exophthalmos and a glaucoma secondary to the exophthalmos.

Following admission the patient was started on pilocarpine (two percent) drops, one to each eye every four hours, and he has been maintained on this medication since admission. A basal metabolism rate on February 25th was reported as plus nine percent. X-ray studies of the skull and the orbits were reported as:

"There is no evidence of any gross osseous abnormalities. There is no evidence of increased intracranial pressure and the sella turcica is smooth in outline and within normal proportional dimensions. The orbital fossa appears to be intact. Both optic foramina are outlined and show no evidence of any erosion to suggest an expanding lesion."

The patient has been followed daily in the eye clinic and is being worked up on the medical service. At present he is undergoing iodine-uptake studies. With his daily observation in the eye clinic the tension in both eyes has ranged considerably from a low of 29/21 mm.Hg to a high of 60/42 mm.Hg, despite the fact that pilocarpine has been continued.

Clinically the exophthalmos has not appeared to improve or become worse; however, he has shown daily changes in the amount of lid edema and conjunctival chemosis. One day there will be a moderate amount of edema and the next day he will go back to his appearance on admission. This has changed repeatedly from bad to worse and then back again.

The patient has also noted extreme variation in visual acuity from the marked visual loss at the time of his admission; vision on March 2nd with correction was R.E., 20/60; L.E., 20/40; on March 7th acuity had gone back to 20/300 in both eyes. The medical service confirms a thyrotropic origin for the condition and has decided to initiate the following therapeutic regime:

The patient was started on 400 mg. of intramuscular cortisone daily and 120 units of ACTH in gelfoam. On this medication the eye complaints clinically improved both subjectively and objectively. There was marked diminution in the amount of conjunctival edema and chemosis and, although the exophthalmometer reading showed only a one-mm. drop in the exophthalmos, the patient subjectively appeared to be markedly improved. The patient's vision returned to 20/25 with correction in both eyes. The tension was normal with the subsidence of the conjunctival edema. This high dosage of cortisone and ACTH was maintained for approximately 10 days, following which the dose was gradually reduced, followed by a slight increase in the conjunctival edema. He was started on thyroid, gradually increasing the dosage up to 1.5 gr. daily. On this medication the patient apparently held his own as far as his eye complaints were concerned. He was discharged to return in one month for recheck and was told to continue with his thyroid daily. He was also given cortone drops to be applied locally to each eye.

The patient returned to the hospital in approximately one month with marked recurrence of the malignant exophthalmos which had been present for two weeks. His vision has decreased to 20/400 in both eyes. Tension was 33 mm. Hg, O.U. There was marked conjunctival edema and exoph-

thalmos. The patient apparently had had a coronary insufficiency attack which may or may not have been associated with his thyroid therapy. He was again placed on ACTH and cortisone and the possibility of pituitary radiation was more seriously considered.

Recurrent pterygium

Dr. Henry G. Farish presented a 46-year-old white woman, who was first seen in the eye clinic of the John Gaston Hospital on August 3, 1953. Her complaint at that time was an extensive recurrent pterygium with symblepharon, O.D. She had had five pterygium operations by two private oculists since the late 1940's; the present lesion was the sixth recurrence. She was sent to a private radiologist by the second oculist but it could not be determined if the radiation administered to the right eye was X rays or beta rays. There were approximately 12 visits to the radiologist.

On the first admission the clinic notes state that there is a pterygium "which covers a vast portion of the cornea," and "the internal canthus is markedly closed since the lids are attached together somewhat, and there is apparently some uniting of the lids with the conjunctiva of the eye itself."

Visual acuity was recorded as: O.D., 20/70; O.S., 20/25. She could read J5 with correction and J8 without correction.

On September 2, 1953, she was admitted to hospital where the pterygium was removed and a mucous membrane graft applied after separation of the symblepharon. Following the operation she did very well until October 29th when more scar tissue formed on the cornea. This was cut and rubber tissue was sewed into the upper fornix. It was finally removed a few days later because of corneal abrasions. On November 19th, vision in the right eye was recorded at 20/200.

It was noted on February 4, 1954, that the pterygium was recurring. One week later it was excised from the cornea and turned up into the upper fornix where it was sutured with 5-0 silk. At this time 50 mg. of beta radium were applied to the cornea for three minutes. This was followed, in the subsequent two months, by a total of 23 minutes of beta radiation given at weekly intervals.

Throughout this period an increasing opacity of the lens in the right eye was noted. On August 5, 1954, her vision was 10/200, O.D., and she was experiencing difficulty with peripheral vision. All beta radiation was stopped after May.

A cataract extraction was advised and she was operated upon August 27th. The extraction was very difficult and was extracapsular through a complete iridectomy. The lens dislocated immediately after grasping with the capsule forceps and threatened to reclinate into the vitreous. It was finally necessary to use a spoon to deliver the lens. As the lens was delivered there was a small loss of vitreous. All of the capsular remains could not be removed.

The eye did not heal well postoperatively and finally dehisced to the extent of about three mm. at the 2-o'clock position. On October 1st, a resuture was performed with three 6-0 chromic sutures and without a flap. It was thought that a flap would be inadequate and could not be well dissected because of the previous multiple surgical traumatism in the area. This repair did not last long and the wound had again dehisced on the fifth postoperative day. She was again returned to the operating room and this time a conjunctival flap was dissected, with difficulty, from the 10- to the 4-o'clock positions. With these maneuvers an opaque gelatinous material, which had the appearance of capsule remains, mixed with vitreous, was expressed from the gaping area. The corneoscleral incision was again sutured, this time with three 6-0 silk sutures. The flap finally retracted and the suture line held. She was discharged to the clinic on October 20th with vision in the right eye down to hand movements. At no time was there any great

degree of inflammatory reaction. The anterior chamber was always deep, even when the incision was open.

Since discharge from the hospital she has been followed frequently in the clinic. There is a definite cleft remaining in the area of the incision which has the appearance of an iris prolapse but, under the slitlamp, is definitely not a prolapse.

She returned for routine check up on November 29th and it was noted at that time that she had an affection of the left eye. There was minimal inflammation but the posterior surface of the cornea was covered with mutton-fat keratic precipitates. The fundus was indistinct because of vitreous haze. At this time vision was: O.D., light perception only; O.S., 20/50.

On dilating the left pupil the remains of posterior synechias were noted on the surface of the lens. She was admitted with a tentative diagnosis of sympathetic ophthalmia. However, specific treatment was withheld except for atropinization until tuberculous involvement was eliminated by the PPD test, which was negative in the first strength after 48 hours. ACTH was then given (20 units, twice daily) for one week and 20 units a day for the next week. Cortisone drops every two hours were prescribed. The keratic precipitates cleared quickly as did the vitreous haze, to a large extent. Vision in the left eye is now 20/40 −2 and she can now count fingers at two feet, O.D. There is still staining of the scarred incision area. The intraocular pressure is 32/19 mm.Hg and there does not appear to be a fistula into the anterior chamber, as tested with fluorescin.

The diagnosis on the right eye was: (1) Recurrent pterygia with symblepharon; (2) radiation cataract; (3) cataract extraction, extracapsular, with failure of the corneoscleral incision to heal, and dehiscence.

The differential diagnosis for the left eye was: (1) Sympathetic ophthalmia; (2) anaphylactic reaction to lens protein of the right eye; (3) tuberculous uveitis.

Retinal cyst

Dr. Alice R. Deutsch presented the case of Mr. O. H. who was seen for the first time in March, 1951. At this time his fundi were normal and his vision 20/20, J1, with correction.

He returned in March, 1953, stating that he needed a change in his glasses. At that time his left eye was unchanged. His distant correction equalled +0.5D. sph. ⊃ +0.5D. cyl. ax 20°, identical with the one in 1951, with an add of +2.00 he saw J1. The right eye could only be corrected to 20/70 and J12. The anterior segment of the right eye was normal and the media were clear. The disc margins were mildly blurred. The macula region appeared very swollen, grayish and granulated, and separated toward the rest of the fundus by a sharp oval glittering line. A large relative central scotoma was present. No metamorphopsia or micropsia was complained of. A tentative diagnosis of central angiospastic retinopathy was made and vasodilators (nicotinic acid, 50 mg., three times a day) were prescribed. Mr. H. was advised to have a complete physical examination and to be treated for a severe alveolar pyorrhea.

He was not seen or heard from until January 16, 1954, when he reported that he could now read without lenses which he had not been able to do for 15 years. He had had a physical examination which was negative. He also had seen his dentist. His left eye again was unchanged. The right eye needed a correction of −4.0D. sph. ⊃ +0.75.D cyl. ax. 170° as compared with his former correction (+0.5D. sph. ⊃ +0.5D. cyl. ax. 180°). With the new correction he saw 20/30+2 and read J3 without correction at about 25 cm.

The anterior segment was normal and the media were clear. No remarkable refractive difference was visible in the center and periphery of the pupil, when the pupil was dilated, but the shadows in the periphery appeared very irregular. When the lens was examined with the slitlamp, the central in-

terval appeared moderately opaque and the posterior Y stood out clearly. The planes of discontinuity were accentuated, especially the anterior surface of the adult nucleus. Otherwise the cortex was clear except for some scattered dotlike opacities.

The disc was normal on funduscopic examination and the arteries normal for the patient's age. The macular region in an area of about 1.0 by 1.5 disc diameters seemed to be swollen but crystal clear. The normal reflexes were obliterated and a broad glittering band separated this area from the rest of the fundus, which was normal. At this time the lesion was thought to be either a flat detachment or a retinal cyst.

When the fundus was examined under the Hruby lens, a large clear cleft was visible in the central retina, broadest in the center of the lesion and gradually attenuated toward the edges; the internal limiting membrane was easily and distinctly visible in the optical section and the external retinal layer appeared as a hazy but transparent stripe in front of the chorioidal band.

The diagnosis of either a large flat intraretinal cyst or a partial retinal schisis was therefore confirmed. The apparent preservation of the neuroepithelium explains the good visual acuity and the absence of a central scotoma, a fact frequently mentioned in fundus changes after central angiospastic retinopathy. It may be questioned whether the change in refraction was caused by the change of the refractive index in the retina itself or whether it was due to beginning nuclear sclerosis which was undoubtedly present.

Daniel F. Fisher,
Secretary, Eye Section.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

December 15, 1954

The 421st meeting of the New England Ophthalmological Society was held at the Massachusetts Eye and Ear Infirmary, Boston.

The afternoon session consisted of the presentation of several interesting clinical cases followed by a pathology conference conducted by Dr. Taylor Smith of the Pathology Department. Dr. Smith presented slides and reviewed the clinical histories on a number of patients with retinoblastoma who had received several different types of treatment. It was his conclusion that no one type of therapy is a solution to the problem and that, even in cases where X-radiation, diathermy, and nitrogen mustard have all been used in treatment, there have been many failures. However, in summary, he expressed the opinion that, in our present state of knowledge, some combination of these three forms of treatment offers the best possibility of helping the patient to survive.

Following the business meeting at the evening session, two papers were presented.

Cavernous hemangioma

Dr. William Stone, Jr., of Boston, presented a report on a patient with a cavernous hemangioma of the orbit which had been removed by exploration of the orbit from a conjunctival incision. Dr. Stone described the approach that he used, reflecting the conjunctiva from the temporal area and retracting the globe nasally, thus exposing the muscular cone. The orbital tissue was held out of the way of the surgeon by use of Serrefine clamps and an excellent exposure of the area resulted.

Dr. Stone described the ultimate removal of the tumor mass by means of compressing the blood from the substance of the tumor and dissecting it out in its capsule. He had opened the orbit the day before for biopsy of the tumor. A clean removal was obtained and the orbit was easily closed. Dr. Stone expressed the opinion that by using the approach described, he had avoided the need of using a Krönlein approach with its attendant technical difficulties.

Discussion. Dr. Virgil Casten pointed

out that the approach described is one which many ophthalmologists have found useful for benign lesions in the orbit. In his experience this type of tumor is one of the most satisfactory from the surgeon's point of view and that, when an anterior orbital approach, such as described in the paper, is used, the surgeon could generally anticipate encountering little difficulty. He reviewed the various types of treatment for hemangioma, concluding that surgery is better than sclerosing solutions, X-ray therapy, or diathermy.

Functioning and nonfunctioning blebs

Dr. Peter C. Kronfeld, Chicago, spoke on this subject. An abstract of his paper follows:

Opinions concerning the mode of action of the so-called filtering operations are still very much divided. In a good many of their cases, Devoe (Tr. Am. Ophth. Soc., **48**:119, 1950) and Meyer (Am. J. Ophth., **35**:788, 1952) questioned the applicability of the theory of fistulation and suggested a neurovascular mechanism as the basis of the normalization of tension that had occurred after iridencleises or sclerectomies.

Sourdille (Bull. Soc. franç. ophtal. **65**: 179, 1952) reported close correlation between filtration and success of iridencleises, on the one hand, and between absence of filtration and failure of the operation, on the other. R. Weekers, a key figure in this field, stated in a number of earlier publications (1948-1951) that the tension-lowering effect of the iridencleisis did not require fistulization. Particularly pertinent to the present topic is Weekers' remark that the persistence of a small focus of conjunctival edema after iridencleises did not demonstrate with certainty that this area is a site of real filtration.

Neurovascular changes in the uvea were assumed to be the principal tension-regulating factor until the advent of tonography and fluorometry made more quantitative analyses of the fluid exchange of the human eye possible. These methods, in the hands of Weekers, as well as in the hands of other investigators, showed clearly that the iridencleisis in chronic glaucoma normalized the ocular tension not by changing the rate of flow through the anterior chamber, but by lowering the resistance to outflow (by raising the coefficient of outflow). Weekers admitted the possibility of filtration into the subconjunctival space where the operative scar was bulky and distended by liquid. Frequently, however, in his cases, the surgical scar was small and flat and the ocular tension strictly normal. In these cases Weekers suggested the possibility of a favorable, resistance-lowering effect of the iridencleisis upon the natural outflow channels.

Best suited for the study of the problem at hand are chronic glaucomas with known flow characteristics before and after the surgery. It usually takes several months before a fairly steady-state with regard to the ocular tension and the coefficient of outflow is reached. Narrow-angle (angle-closure) glaucoma or secondary glaucomas are not suited for the purpose of this study since most of them have a very considerable outflow facility through their natural channels, making the outflow through other channels only a fraction of the total. In the presence of a very considerable outflow through natural channels a small amount of flow through surgically produced channels is difficult to demonstrate.

In presenting his findings in chronic glaucomas the speaker followed a classification based upon biomicroscopic characteristics of the external site of the operation, similar to Pillat's original classification. Group or type one in this classification considers the blebs or filtering cicatrices in the strict sense, that is localized anemic, partly dense and partly thin-walled and thin-partitioned, multichambered structures. Type two has the diffuse, milky, pitting edema of the conjunctiva that many observers seem to have overlooked. Type three is characterized by crystal-clear thin conjunctiva stretching tautly over the clearly visible exteriorized piece of iris. Type four bears a superficial

similarity to type one in that there is a localized elevation which, in contradistinction to type one, is solid and as well as or better vascularized than the surrounding conjunctiva. Under type five are classified the external appearances characterized by small, flat, or slightly elevated opaque areas the nature of which, edema or scarring, cannot be determined by biomicroscopy alone. There exist mixtures of type one and two, transitional forms between two and three and one and four.

Blebs of type one are present in a large majority of truly successful, that is, completely tension-normalizing, iridencleises or sclerectomies. Biomicroscopically as well as histologically these blebs are made up of mixtures, in greatly varying proportions, of dense fibrous areas, on the one hand, and delicate cystic, thin-walled and thin-partitioned areas, on the other. The latter areas are concerned with the function of the bleb since their degree of development closely parallels the lowering of the tension. The coefficient of the facility of outflow in blebs of type one varies from 0.15 to 0.6, the higher coefficients being associated with lower or very low tensions. Transconjunctival passage of aqueous can be demonstrated by placing filter-paper discs or other absorbent materials over the thin-walled portions of the bleb. Rarely one finds blebs of type one associated with insufficient lowering of the tension. Close biomicroscopic examination of these cases reveals only small, thin-walled areas and usually no internal, gonioscopically visible opening.

External appearance of type two is probably the ideal operative result. The ocular tension is usually lowered and the coefficient of outflow raised to the normal level. No transconjunctival transfer of aqueous is demonstrable. The subconjunctival filtration of aqueous may be made more conspicuous by external pressure applied against the lower half of the globe,

External appearance of type three after iridencleises in cases of chronic open-angle glaucoma is associated with failure of the operation, that is no lowering of tension or raising of the co-efficient of outflow. If the anterior chamber was absent for several days after the operation, the outflow may actually be worse than before the operation.

External appearances of type one, two, one plus two, three, and four make up 92 to 93 percent of all the operated eyes. There remain seven to eight percent of operated eyes in which the external appearance is that of type five or, perhaps, that of the borderline condition between one and four and three and five. In some of these cases the operation has definitely resulted in normalization of tension and improvement of outflow. In such cases the presence of subconjunctival filtration is suggested by a test (Kronfeld and McGarry, Tr. Am. Ophth. Soc., **48**:107, 1951) which consists of determining the drop in ocular tension caused by application of external pressure by means of a dynamometer, to several different portions of the globe. Application of the dynamometer to the operative site interfers with the filtration through that area and therefore is followed by a smaller drop in tension than application of the same amount of external pressure to other sites on the surface of the globe. The speaker asked that this test be used in cases where the presence of subconjunctival filtration cannot be conclusively demonstrated by biomicroscopy alone.

Transconjunctival or subconjunctival filtration is suggested by a relative insensitivity of the outflow to conditions exemplified by Valsalva's experiment. This experiment causes in normal eyes a very sharp rise in tension which is usually very slight in eyes with functioning blebs of type one, two, or five.

Discussion. DR. PAUL CHANDLER pointed out that in DeVoe's series which Dr. Kronfeld had cited, Dr. DeVoe had no record of the type of glaucoma he was dealing with, and that when trephination is done for narrow-angle glaucoma there is no need for filtration, since the iridectomy cures the patient. He added that there are very few

patients with open-angle glaucoma who fail to show evidence of filtration and still have normal tension and normal tonographic measurement of outflow. However, he mentioned one of his own patients who has had bilateral trephinations who does not show any clinical evidence of filtration from objective examination of the appearance of the eye, and yet the patient has normal tension in both eyes with normal tonographic measurements. He added that this patient had had a preoperative tonographic workup that had definitely shown the outflow to be in the range of glaucoma. He thought that this indicated some mechanism of filtration had been established, although there was no bleb and no other clinical evidence of filtration. He went on to state that, in regard to the theory of a neurovascular mechanism to cure glaucoma—"Fie on it"! He cited numerous cases of narrow-angle glaucoma in which operations have been done and failures resulted, all of which showed inadequate operative procedures. The failures were the result of not making complete holes in the iris in these narrow-angle cases. He added that he agrees with Dr. Weekers, that is, "no drainage, no cure"; never mind the neurovascular reflexes.

Dr. Morton Grant commented on the Valsalva experiment described by Dr. Kronfeld, raising the question as to whether the rise in tension was not due to engorgement of the vascular bed in the eye rather than obstruction to outflow because the tension rises so fast in this experiment; and whether the failure of the Valsalva test to show a marked rise in pressure, in patients who have had filtration surgery, might not be due to the fact that the initial tension was lower in these patients. If such were the case, the same increased volume within the eye might produce less effect on the pressure of the eye than if the initial pressure were higher. He asked Dr. Kronfeld two questions; the first, whether Dr. Kronfeld had observed any evidence to indicate that the aqueous veins gradually close following a trephination or other filtration procedure, and that, when this happens, if the filtration operation itself fails, the glaucoma would be worse than before. (He cited a theory of Dr. H. Goldmann as the background for this question.) The second question was whether Dr. Kronfeld had made tests for aqueous outside of the eye following cyclodiathermy procedures, because in some of the patients at the Massachusetts Eye and Ear Infirmary, there were some definite blebs formed over these surgical wounds which seemed to indicate evidence of filtration.

Dr. Kronfeld agreed with Dr. Chandler that too many surgeons today are striving for a minimum sized iridectomy and, as a result, have ended with a partial iridectomy in which the iris has not been completely punctured. In reply to Dr. Grant's discussion, he stated that in his Valsalva experiments the initial tension had been the same in the controls as in the tested subjects, one of the controls being himself where the tension normally runs around 16 mm. Hg. He expressed the opinion that the Valsalva test does indeed indicate the patency of outflow channels. In reply to Dr. Grant, Dr. Kronfeld stated that, in his opinion, the normal outflow channels usually remain patent and that, in these patients, even with filtering blebs present, aqueous veins can be observed. He feels that the filtration which occurs following surgery is a combination of that which normally takes place through the iris meshwork into the canal of Schlemm, assisted by the filtering bleb as a result of the operation. He states that this is particularly true in patients in whom the chamber has been formed most of the time; that is, when the postoperative flattening of the chamber is minimized. In reply to the second question, he stated he had never personally made any observations on outflow following cyclodiothermy, although he thought Dr. Grant's observations on this question were quite interesting.

David H. Scott,
Recorder.

YALE UNIVERSITY CLINICAL CONFERENCES

February 11 and February 25, 1955

Dr. R. M. Fasanella, *presiding*

Management of reading problems

Miss Josephine E. Williams, M.A., reading consultant, New Haven Board of Education, and Mr. John Duggan, B.A., reading consultant, Yale University, spoke on this subject.

Miss Williams: Many of our children become reading problems right from the first grade. This is explained by the fact that one third of the first-grade class is not ready to read. To evaluate the readiness of first-graders to read, tests are given at the end of kindergarten. Intelligence alone is not sufficient. Anything which stops a child's concentration prevents his reading. Those children who are judged not intellectually or psychologically ready to read are separated in order not to force them to do something for which they are not ready. There are usually more boys who become reading problems than girls, because the boys are not as emotionally mature as girls of equal age.

Miss Williams then demonstrated how the reading act is first learned. She made a new set of symbols to represent words with which the audience was well acquainted. This was equivalent to a child learning to read. The child's difficulties were thus well illustrated. The tachistoscope was demonstrated to show how mechanical devices could be used to teach new fixation habits which would be carried over to normal reading.

Mr. Duggan then explained why it is necessary to teach reading on a college level. The variation in reading speed in the usual Yale undergraduate freshman class varies from 125 to 700 per minute on the same easy material. It is felt necessary to increase the speed of the slower ones. Mr. Duggan then explained why some students read slowly and get satisfaction from doing so. He then very briefly explained that he used the telebinocular on each student and suggested appropriate professional eye examination if any problems were encountered.

The aim of the half-year course is to teach the student flexibility, to vary reading speed with the material. Speed with comprehension was stressed, not speed alone. To show that the course was fulfilling its purpose, Mr. Duggan stated that the grades of the students had become better and some students who would have failed without the increase in reading speed were enabled to maintain satisfactory grades. A short movie than demonstrated some points.

Discussion. Dr. C. C. Clarke discussed the part of the ophthalmologist and orthoptic technician in the management of reading problems. He explained why he thought it was the job of the ophthalmologist to investigate those persons who are reading problems and not the job of the reading instructor to give such tests as the telebinocular and orthorater. Having the layman administer these tests, which are merely for screening and not for diagnosis, just gives a scientific aura to the suggestion to see an oculist. In addition to referring all reading problems, schools should refer children who have poor visual acuity at near or far or who complain of ocular symptoms.

Miss Derdell (social service) agreed that it was best not to intimidate parents with machines in order to get them to the ophthalmologist.

Dr. Louise Lovekin stated that she felt that even small hyperopic and astigmatic errors should be corrected if the patient has symptoms.

Plastic repair of ocular injuries

Dr. Arthur G. DeVoe first re-emphasized three principles he had mentioned on another occasion.

1. The division of the lid into two essential layers; the skin-muscle layer and the tarsal conjunctival layer.

2. Recognition of limitations when no

further benefit will result from additional surgery.

3. Proper time of operation. Retention of function is the first consideration, plastic improvement secondary. Repair should, in general, be done as early as possible, but infection may have to be treated first. There is a question as to the handling of an injury case 12 hours old when first seen. One may, under certain circumstances, wait to see whether fulminating infection is developing before proceeding, though this may not be the usual approach.

In lacerations across the upper lid, one should try to identify the levator and suture it separately. He disregards the upper canaliculus, but makes an attempt to repair the lower canaliculus. In his experience, the use of polyethylene tubes has not been very satisfactory. In many cases, either an abscess has developed or the tube spontaneously is extruded after two to four months. However, he would still try repairs with stilet and suture.

In burns, the retention of vision is paramount. Immediate grafting not advisable around the eye. In many cases, the lid swelling itself forms a satisfactory pressure dressing to the cornea, and no further dressing is needed. The opinion of many general and plastic surgeons now is to leave facial burns practically untouched.

The use of EDTA (ethylene diamine tetra-acetic acid) salts in the treatment of lime burns and corneal calcification, as in band keratopathy, was described. All epithelium is curetted, then a solution of the sodium salt of the drug is applied to the cornea in an iontophoresis tube or similar applicator. An 0.05 molen or 1.8 percent solution is used. In severe lime burns, this can be applied immediately to remove calcium. In the older cases of band keratopathy, it will remove calcium plaques, but not fibrous scar.

Two methods of treating trichiasis were shown:

1. Transposition of two long horizontal strips of skin, one with the cilia. In the discussion, it was mentioned that this seemed contrary to the usual rules for pedicle flap viabiilty. Dr. DeVoe agreed, but felt that here the strips were acting essentially as free grafts.

2. Removal of a V-shaped wedge of tarsus was described.

Epicanthus in the more severe grades is a difficult problem. Most of the less severe cases need no surgery. Dr. DeVoe thought some modification of the Z-plasty was best. The V-Y procedure has been generally discarded, but may possibly be of use in blepharophimosis. The Blair-Brown procedure, which is a form of double Z-plasty, seems satisfactory.

Dr. DeVoe described his experience thus far with peritoneal grafts in cases where all conjunctiva is lost. This occurs not infrequently in Stevens-Johnson syndrome. In the first such case, when the lids were cut apart six weeks postoperatively, shrinkage occurred and the implant was extruded. Now he has in one case sutured the lids over a conformer and plans to leave it closed for six months. Reports from elsewhere, thus far, have not been enthusiastic.

William I. Glass,
Recording Secretary.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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MARFAN'S SYNDROME*

The condition originally described by Marfan[1] as dolichostenomelia was apparently a congenital abnormality of the skeleton characterized by the length and slender proportions of the long bones. The alternative title of arachnodactyly was introduced by Achard[2] to emphasize the "spiderlike" appearance of

* Reprinted by permission from *The Lancet,* May 21, 1955, page 1062.

the hands. The syndrome is now taken to include various ocular abnormalities, of which subluxation of the lens is the commonest, and cardiovascular lesions, of which aortic dilatation and dissecting aneurysm are probably the most usual and are certainly the most serious.

Achard first drew attention to a hereditary and familial tendency, but according to Rados[3] the family history was positive in less than 40 percent of cases. McKusick[4] has now re-emphasized the importance of heredity in the genesis of the syndrome. An inquiry into the kinships of 50 unequivocal cases revealed a total of 105 affected persons, excluding many doubtful cases. Of the relatives of all the available patients with subluxation of the lens, 70 percent had recognizable stigmas of the syndrome. The genetic data indicate that the disease arises as a result of a single mutant gene which is subsequently transmitted as a dominant. McKusick concludes that about 15 percent of all cases arise from such a mutation and 85 percent by transmission.

The large number of tissues and structures that may be affected in Marfan's syndrome suggests that, if a single defect is responsible, this must be fundamentally important. Its nature is by no means clear; from the microscopic appearance of cystic medial necrosis, which is the usual finding where the aorta is dilated or has undergone aneurysmal dissection, McKusick concludes that the elastica is probably the tissue primarily affected. But it is difficult to see how such involvement can be responsible for the various ocular and skeletal abnormalities "unless one assumes that the presence of this defect during embryogenesis produces an abnormal setting in which these particular anomalies occur with increased incidence."[4] In any event the fundamental defect is functional rather than structural, at least in so far as it affects the aorta. What is inherited is apparently an inability of the elastic tissues to withstand the normal wear and tear of life. Consequently the patient may in childhood show no evidence of cardiovascular abnormality, but in adult life have severe aortic dilatation, valvular incompetence, and myocardial hypertrophy.[5]

The presence of an underlying biochemical defect is strongly suggested by the production in rats fed on the seeds of Lathyrus odoratus of a syndrome that includes skeletal defects and degeneration of the media of the aorta leading to dissecting aneurysms.[6] An active principle recently extracted from the seeds of L. odoratus is β-amino propionitrile. Lathyrism in rats cannot justifiably be regarded as a phenocopy of Marfan's syndrome. But the striking similarity of many of the features of these two conditions does support the view that the complex anomalies that make up the syndrome in man are based on a single transmissible biochemical defect.

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CORRESPONDENCE

"First, do no harm"

Editor,
American Journal of Ophthalmology:

I should like to comment on the fine symposium in the August, 1955, issue of The American Journal of Ophthalmology regarding retrolental fibroplasia, and on Dr. Reese's accompanying epitaph for the same disease. I cannot let this opportunity pass without perhaps remarking that American medicine and the various people most directly interested have done an extraordinarily good job in fighting an unnecessary fire. Retrolental fibroplasia is certainly a man-made disease. That it has been made by man's thought-

lessness is perhaps a somewhat too harsh verdict, but the disease itself is a violation of the old principle of "First, do no harm." It is perhaps fitting that this condition should have arisen in America where civilization is inclined to be somewhat wasteful of its resources, and where the philosophy is rampant that if a little is good, a lot more is wonderful.

We chide our patients for taking twice the prescribed dose. We blast them if they take a lot of harmful and needless medications, and we become very disturbed when they poison themselves from any miscarriage of self-therapy.

Yet here we are faced with a confession of having ignored one of the oldest doctrines in medical care and, in an effort to save the lives of many premature infants, have gone hog-wild on oxygen therapy without considering the possibility that there might be some specific dangers to it.

In many ways we may be creating a similar deluge for ourselves with wanton antibiotic therapy, and some of the other newer and as yet ill-defined therapeutic measures which we now have in our hands. It is incumbent upon us all then to profit from the experience of retrolental fibroplasia, and it should give us pause in the management of any new therapeutic tool.

(Signed) W. A. MacColl, M.D.
Seattle, Washington.

BOOK REVIEWS

RELAX AND SEE. By Clara A. Hackett. (With Lawrence Galton.) New York, Harper & Brothers, 1955. 300 pages. Price: $4.00.

P. T. Barnum made a comfortable living on the theory that there's a sucker born every minute. It seems evident that even in the United States—which owes its great material wealth, its low death rate, its low maternal mortality rate, its low rate of blindness, and its long span of useful, productive life to the accomplishments of the physical and biological sciences—there is still as great a market for mumbo-jumbo as in the most primitive parts of Africa. Clara Hackett claims that she has brought about vast improvements of visual acuity in 90 percent of 2,800 persons who have had such varied conditions as myopia, cataracts, glaucoma, optic-nerve atrophy, conical cornea, and macular degeneration. Although she states that most of the latter patients, if not all, were at the same time under medical care, by implication she gives credit for any help the patient might have received to her exercises rather than to the medical care which they were getting. One wonders how many of the 40 retinitis pigmentosa cases she reports as improved by her exercises have also been reported as improved by the Filatov treatment—it is not inconceivable that some of these persons, grasping at straws, received both kinds of therapy at the same time.

Possibly one should not begrudge Harper Brothers and the Bates cult the shekels they extract from the gullible. Yet there is obvious danger here to the eyes of our fellow Americans. One shudders to think of the amblyopias which will be produced by having children follow the Hackett-Galton treatment for strabismus, rather than proper care; and of the preventable blindness that will occur among patients with chorioretinitis and glaucoma, if they rely on her exercises rather than on medical advice. According to the publisher's jacket blurb, Miss Hackett apparently has so far evaded the application of medical and optometric practice regulations. She calls her treatments "training," her patients "students."

It was Bates who developed most of the hocus-pocus Miss Hacket recommends, and she acknowledges her debt. She and Mr. Galton, "medical" columnist for Hearst's *Cosmopolitan*, are in error when they ascribe to Bates the idea that visual training can improve sight and when they credit Bates with the "revolutionary idea" that it is the extra-

ocular muscles which control accommodation, rather than the ciliary muscle. This erroneous concept was held by many physicians until early in the 19th century when Thomas Young observed the action of the ciliary muscle in a bovine eye. Other poorly trained practitioners before Bates held to this concept, despite the great strides in ophthalmic science in the 19th and 20th centuries. Such laboratory work on accommodation as he did, Bates carried on largely on fish, and even nonmedical physiologists who have studied accommodation have pointed out the vast difference between the process in fish and mammals.

The idea of visual training, of course, antedates Bates by many years. Most ophthalmologists have been puzzled and have felt frustrated by myopic patients who seem to have a lower degree of visual acuity than is justified by their refractive error. It is recognized that some persons who have blurred vision from various causes fail to use such vision as they have to try to interpret the blurred image they see—they give up at once, rather than use the little cues and tricks which others find helpful. Serious students of visual training have followed the scientific method rather than a sectarian approach. The scientific method involves accurate observation, thorough reporting of all factors which might bear on the results, the setting up of adequate controls so that an hypothesis may truly be tested, and integrity and absence of emotion in recording the findings. Those who have followed the scientific method have developed orthoptics as an aid in the total management of muscle imbalance, inadequate fusion, and the efficient, comfortable use of binocular vision. Although orthoptists have avoided the exaggerated claims of the Bates cult, there is no justification for the assertion in the foreword by Dr. William Gutman, a member of the American Institute of Homeopathy, that the principle of systematic exercise and training has been strangely overlooked in relation to the visual function.

It is significant that Hackett and Galton ignore the findings of the Johns Hopkins and Washington University investigations of the claims for visual training in myopia. It is true that instead of devoting their years of preparation to a servile study of Bates, ophthalmologists study the function, chemistry, and microscopic anatomy of the normal and abnormal human eye; they learn to observe the function of the inner eye through the retinoscope, ophthalmoscope, slitlamp biomicroscope, and gonioscope. Not always trusting their own observations, they make photographs of what they see and call on other disinterested scientists to check their findings. Possibly, Hackett and Galton see no merit in this kind of approach and therefore attach no value to ophthalmologic investigations of the results of visual training. On the other hand, I feel in studying the Hackett modifications of Bates' system—sunning, palming, the long swing, the lazy daisy swing, edging, blinking, and so forth—much as neuropsychiatrists must feel when asked to review works by phrenologists, or astronomers the writings of astrologers. In October, *Coronet* ran an article by Galton based on the Hackett exercises, and followed the article with a page of barbed comments, by Oscar Wilde, among which was this reflection on *Homo sapiens*, "I can believe anything—provided it is incredible."

Franklin M. Foote.

KERATOPLASTY. By R. Townley Paton, M.D. New York, McGraw-Hill Book Company, Inc., 1955. Cloth bound, bibliography. Price: $28.50.

The advent of antibiotics, eye-banks, and improved surgical instruments has changed corneal transplantation from a mystifying, dramatic procedure done by only a few ophthalmologists to a practical ocular operation which can be done by any interested, well-trained eye surgeon. Dr. Paton's timely book will be a great aid to all ophthalmic surgeons

interested in such procedures.

The text is divided into 13 chapters and is headed by a very interesting *Foreword* written by Dr. Derrick Vail. The subject matter includes the history of corneal grafting, the anatomy and physiology of the cornea, case selection and prognosis, selection of donor material, pre- and postoperative care of the eye, surgical technique, and the pathogenesis and treatment of postoperative complications. The material in each of these sections is documented by a well-chosen but representative bibliography.

The main value of this book is the comprehensive, unified review of a surgical procedure which has been crystallized into a practical clinical approach during the past decade. Such a text could only have been written by a single author who had spent many years in preparation for his publication.

It is difficult to single out an individual chapter as the high point in this presentation, but the chapters on case selection and donor material should be read by all ophthalmologists who are interested in either referring patients for corneal transplantation or in performing this procedure. It is also difficult to be critical of this work except to mention that while the color prints of preoperative and postoperative eyes are nice to look at, they add to the cost of printing and thus make this a relatively expensive book.

A. Edward Maumenee.

History of Medical Practice in Illinois: Volume II, 1850-1900. Edited by David J. Davis, M.D. Chicago, Illinois State Medical Society, 1955. 530 pages, index. Price: $10.00.

The Illinois State Medical Society was organized in 1840 and its hundredth anniversary was celebrated in a centennial issue of the *Illinois Medical Journal* in May, 1940, in which the varied contributions of Illinois physicians to medical advance were reported. The initial volume of this more elaborate presentation was edited by Dr. Lucius H. Zeuch and published in 1927. The series record not only professional progress but also the wider role played by Illinois physicians as citizens. Evanston was named after Dr. John H. Evans who was instrumental in founding Northwestern University; and its Davis Street was named after Dr. N. S. Davis. The Governor inaugurated in 1857 was Dr. W. H. Bissel. Prior to 1850, the Illinois Country was characterized as "a land of pestilence" and Chicago was described as "one chaos of mud, rubbish and confusion." The pioneer had to be hardy to survive.

After 1850, the several specialties developed rapidly and, by 1900, were well defined. Dr. William A. Mann of Northwestern University Medical School contributes a delightful chapter of 30 pages on the ophthalmology of this period with fascinating details about its major and minor personalities. The first paper on eye disease in the *Transactions* of the Illinois State Medical Society was on the use of collodion in entropion by Dr. E. S. Cooper. Dr. Cooper, who was primarily a surgeon, moved to the West Coast and the Cooper Medical College, now the Medical Department of Stanford University, was named in his honor in 1888. In 1880, Dr. Everett discussed the discovery of homatropine by Ladenberg of Germany. Dr. Thomas Hall Shastid, who contributed some 3,000 articles to the American Encyclopedia of Ophthalmology, practiced in the Illinois towns of Pittsfield, Galesburg, and Marion before moving to St. Louis and Duluth. The distinguished editor of this volume, Dr. D. J. Davis, died suddenly on December 19, 1954. In the chapter on medical geography in Illinois he makes the interesting observation that the Illinois River marked the upper margin of the trachoma region in this state.

James E. Lebensohn.

Archives of the Ophthalmological Society of Northern Greece. Thessaloniki, Volume III, 1954. 172 pages.

Polychronakos and Leanis report the results of 200 cases of cataract extraction from

the eye department of the Municipal Hospital of Salonica. The corneal section is done with a Graefe cataract knife without conjunctival flap. Three corneoscleral sutures are used and, after a peripheral iridotomy, the lens is extracted with the Arruga forceps. In 81 percent of the cases, the lens was delivered intracapsularly and only six patients developed complications postoperatively. Polychronakos also presents a case of traumatic dislocation of the lens under the conjunctiva caused by a bull's horn, and reports the results of five plastic operations for epithelioma of the lower lid and cicatricial entropion. The same author and Anastassiadis present a case of recurrent mixed tumor of the lacrimal gland.

C. Konstas presents a case of group pigmentation of the retina and another of choroidal nevus; he recommends the application of irgapyrin in uveitis and posttraumatic conditions of the globe. He observed a case of temporary retinal ischemia in a hypertensive patient. George Konstas reports a case of metastatic endophthalmitis in a 70-year-old man with bronchopneumonia.

Tsopelas summarizes the present conceptions of the pathogenesis and treatment of sympathetic ophthalmia, reviewing Schreck's work on rickettsia sympathetica and recommends fever therapy as most effective. Georgiades presents a case of sympathetic ophthalmia and his findings support Schreck's theory of the pathogenesis. He observed iris nodules in a case of neurofibromatosis and reports a case of unilateral Mikulicz's syndrome. He also presents the X-ray findings of 40 patients with Fuchs' syndrome (cataract, heterochromia, corneal precipitates); 23 of them had changes in the cervical vertebras. The same author reports five cases of a new syndrome consisting of hyperchromia of the iris with cataract. He believes that this is a separate entity, differing from the other two similar conditions—hypochromia cataract and Fuchs' syndrome. Histologic examination of the iris in four of the cases showed strong pigmentation, atrophy and sclerosis of the stroma and sclerosis of the vessels. The author believes that the cataract is due to similar vascular changes of the ciliary body. (His extensive monograph on the subject of heterochromia iris has been reviewed separately in THE JOURNAL, 40:446, 1955.) He also reports the second case in Greece of essential atrophy of the iris. With Petrides he reports a case of incomplete mandibulofacial dysostosis and a rare case of large lipomas of both lower lids in a 60-year-old man.

Georgiades and Alexiades report a case of conjunctival parasites contracted following a fall on a heap of grass and two cases of sympathetic ophthalmia treated successfully with antibiotics and pyretotherapy.

Brissimis emphasizes the value of orthoptic training for patients with strabismus who have had surgery and presents the results in six. Bouzas reports two cases of orbital mass which developed after enucleation for retinoblastoma and proved to be a cyst and not a recurrence of the tumor. He emphasizes that no exenteration of the orbit should be performed before biopsy is done.

Zervacakos reports two cases of foreign body of the lens well tolerated and without the development of a cataract.

This volume is well printed and illustrated and contains brief summaries in French and English.

Manos A. Petrohelos.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

5

DIAGNOSIS AND THERAPY

Caselli, Francesco. **Ergotamin-tartrate caffeine (Cafergot) in ophthalmic hemicrania and in hemicrania syndromes secondary to eye conditions.** Arch. di ottal. 59:393-400, Sept.-Oct., 1955.

Cafergot in 10 healthy subjects lowered very slightly the retinal arterial pressure of five and the systolic and diastolic pressure in the humeral artery of seven persons. The same effect was observed in 12 patients with ophthalmic migraine, 11 of whom obtained relief from the hemicrania. In acute glaucoma (6 cases), acute exacerbation of chronic glaucoma (14), hemorrhagic glaucoma (3), and secondary glaucoma in iritis (5), the patients did not obtain any relief. (8 references)

John J. Stern.

Chambers, Arthur L. **The use of cortisone in treatment of chorio-retinal damage resulting from observation of atomic detonation.** Military Med. 117:338-342, Oct., 1955.

Three days after viewing an atomic detonation without glasses, the patient complained of seeing an absolute scotoma in the left eye, the actual shape of the blast. Vision varied from 20/60 to 20/200. A white fluffy exudative lesion was present in the macular area. The patient was given cortisone systemically and on discharge from the hospital, the vision was 20/50. The exudative inflammatory reaction was curtailed by the use of cortisone. (12 references) Irwin E. Gaynon.

Christensen, L., and Rowen, G. E. **Diagnosis of malignant melanoma by subretinal fluid studies.** A.M.A. Arch. Ophth. 54:477-480, Oct., 1955.

The authors feel that aspiration biopsy of subretinal fluid in eyes with suspected tumors is contraindicated because of the danger of extraocular extension. (6 figures, 11 references) G. S. Tyner.

Crick, R., Hoyle, C., and Mather, G. **Conjunctival biopsy in sarcoidosis.** Brit. Med. J. 2:1180-1181, Nov. 12, 1955.

In this preliminary report a rapid and easy method for confirming the diagnosis in patients with probable sarcoidosis is described. Biopsy is done by picking up with a forceps a small conjunctival fold containing follicles and snipping it out with one cut of the scissors. The 2 mm.

incision need not be sutured; it heals completely in a few days without infection or symblepharon. Interpretation of histologic preparations is more difficult than in liver or lymph nodes because the conjunctiva normally contains inflammatory and lymphoid cells. For this reason 50 serial sections are cut and studied in each case. The method has been used in 28 cases of suspected sarcoidosis and the results suggest that the procedure will prove to be of diagnostic value. (3 figures, 5 references) F. H. Haessler.

Degand, Gros, and Sergeant. **The use of streptokinase-streptodornase in a case of tuberculous meningitis with ocular complications.** Ann. d'ocul. **188**:546-555, June, 1955.

Complete bilateral blindness suddenly occurred in a 15-year-old girl with tuberculous meningitis. Fundus examination showed bilateral optic atrophy. Because of the possibility that the atrophy might be due to compression from fibrinous exudate in the optic chiasmal region, an intrathecal injection of streptokinase-streptodornase was given. This was repeated in five days and again in twelve days. Forty-eight hours after the second injection, a pink colour returned to the discs and intense vasodilatation was observed throughout the fundi. Central vision gradually returned to normal, but a permanent homonymous hemianopia remained. Severe side effects (headache, vomiting, pain in the back, and, on one occasion, convulsions) followed the dosages given (5,000 English units; 30 American units). It is possible that comparable therapeutic results might have been obtained with smaller doses. Daily intrathecal injections of small doses (1,000 to 2,000 English units; 6 to 12 American units) might be valuable as a prophylactic in other cases. (7 figures, 14 references) John C. Locke.

Donin, J. F., Henderson, J. W., and Grindlay, J. H. **Evisceration of the eye with implantation of polyvinyl sponge.** A.M.A. Arch. Ophth. **54**:373-380, Sept., 1955.

Polyvinal sponge implanted in eviscerated dog eyes was generally well tolerated in an observation period of one year. The authors believe this substance would be well tolerated in man if hemorrhage could be controlled and infection prevented. (4 figures, 1 table, 17 references) G. S. Tyner.

Esposito, Albert C. **An orbital compressor.** A.M.A. Arch. Ophth. **54**:432-433, Sept., 1955.

A simple device for obtaining hemostasis after enucleation is described. These compressors may be purchased from the Storz Company. (2 figures) G. S. Tyner.

Foster, John. **The use of the Fermin Nash slide rule in the diagnosis of unilateral exophthalmos.** Arch. Soc. oftal. hispano-am. **15**:517-521, May, 1955.

Foster devised a slide rule for the differential diagnosis of unilateral exophthalmos, based on the same principle as the Fermin Nash slide rule for thoracic lesions. He applied this scale to 35 cases of verified orbital tumors. In two cases the scale pointed to two diagnostic possibilities, one of which was correct. In eight cases the diagnosis was incorrect. The total number of correct diagnoses on the slide rule appeared to be equal to those arrived at clinically by the author and the neurosurgeon. Foster envisions the application of such a device to other fields of ophthalmology, as for example the classification of strabismus and therapeutic indications. (2 figures) Ray K. Daily.

Hedges, T. R., and Walsh, F. B. **Optic nerve sheath and subhyaloid hemorrhage as a complication of angiocardiography.** A.M.A. Arch. Ophth. **54**:425-427, Sept., 1955.

Intraarterial angiography was attempted on a 36-year-old patient with congenital heart disease. Death occurred three hours after instillation of 22 cc. of 70 percent iodopyracet into the right carotid artery. Autopsy showed unilateral optic nerve sheath hemorrhage and subhyaloid hemorrhage on the injected side. No intracranial meningeal bleeding was found, thus making it evident that optic nerve sheath hemorrhage can occur independently without blood being forced through the subarachnoid space via the optic foramen. (3 figures, 2 references)

G. S. Tyner.

Kramer, F. **Experiences on the value of hyaluronidase.** Klin. Monatsbl. f. Augenh. **127**:364-367, 1955.

The author adds 25 TRU to 3 cc. procaine and has used it for 500 operations. The resorption of the anesthetic was facilitated and the hypotony of the globe more pronounced. In animal experiments it was found that a subconjunctival injection of hyaluronidase accelerates the absorption and effect of miotics. It did not increase the penicillin concentration in the aqueous after a subconjunctival injection of the antibiotic. (14 references)

Frederick C. Blodi.

Kratka, William H. **Isoniazid and ocular tuberculosis.** A.M.A. Arch. Ophth. **54**: 330-344, Sept., 1955.

Nonallergic immune rabbits and human patients were studied to evaluate the effectiveness of isoniazid in the treatment of ocular tuberculosis. The drug is of low toxicity in therapeutic levels. Experimentally it penetrates ocular tissues well. It is most effective when used in combination with streptomycin and para-aminosalycilic acid. Clinically, patients were treated who showed a positive PPD skin test. In man it was 69 percent effective in cases of posterior uveitis and 52 percent effective in cases of anterior uveitis. Preliminary studies suggest that concurrent use of adrenocorticosteroids may be of help. (20 figures, 2 tables, 55 references)

G. S. Tyner.

Manchester, P. T., and Bonmati, J. **Iodopyracet (diodrast) injection for orbital tumors.** A.M.A. Arch. Ophth. **54**:591-595, Oct., 1955.

The use of this material to outline orbital tumors can be extremely hazardous. Its untoward effects can be somewhat lessened by prior instillation of hyaluronidase. (3 figures, 4 tables, 12 references)

G. S. Tyner.

Montes, Galvez J. **Electronystagmography: technique, applications, and experimental data.** Arch. Soc. oftal. hispano-am. **15**:488-495, May, 1955.

The experimental instrumentarium and technique of electronystagmography is described. It is pointed out, that while the procedure is somewhat complicated, and requires expensive equipment, it does provide a useful and simple method for the objective study and detailed analysis of nystagmus and of ocular motility in general. From a study on cats the author concludes that the electronystagmogram is in probability a composite curve resulting on one hand from a displacement of the electric axis of the eye, and on the other hand from variations in the orbital equilibrium; the muscular contractions do not appear to play an important part. (6 figures, 2 references)

Ray K. Daily.

Valentin-Gamazo, Fernandez Ignacio. **Expulsive hemorrhage.** Arch. Soc. oftal. hispano-am. **15**:613-626, June, 1955.

The literature on the pathogenesis and types of this disastrous surgical complication is reviewed, and the importance of prophylaxis emphasized. Among the measures advocated are a thorough preoperative study of the cardiovascular-renal system, normalization of the ocular ten-

sion, a retrobulbar injection of novocain-adrenalin, slow, gentle and skillful surgery to give the eyeball time to adapt itself to the disturbed physiologic conditions and to minimize the effect of the surgical trauma and general akinesia with curare. Above all, however, the great value is emphasized of the pharmacodynamic potentialization as advocated by Barraquer, in eliminating all the factors predisposing to and provocative of an expulsive hemorrhage. The combination of drugs advocated by Barraquer eliminates the disruption of the equilibrium of the neurovegetative system and the accidents following such a disturbance; it induces general and local tranquility by an indifference to surroundings and to the surgical intervention; and it diminishes the dose of curare necessary to obtain a satisfactory general and local akinesia. (25 references) Ray K. Daily.

6

OCULAR MOTILITY

Appelmans, M., Michiels, J., and Jansen, E. **Ocular muscle anomalies in the presence of tumors.** Bul. Soc. belge d'opht. **109**:53-60, Feb., 1955.

One or more of the extrinsic ocular muscles may be encroached upon by either a primary or metastatic tumor or they might be exposed to considerable stretching when the orbit is invaded by a tumor. Two pertinent case histories are discussed in detail. In one, in a young woman who had disseminated metastasis of a cancer of the breast, no impairment of motility was found in spite of a dense infiltration of all the ocular muscles with tumor cells. In the second case, a 43-year-old woman with an unusually large mucocele of the left frontal sinus had severe displacement of the eyeball forward and to the temporal side. The elongation of the extrinsic muscles was several centimeters; exact measurements were not possible because of the complete change of position of the eye.

Attention is called to the remarkable elasticity and reserve power of the muscles which enables them to adapt themselves to the most complicated anatomical conditions; being of comparatively slightly differentiated tissue, controlled by a very complex innervation, they keep active as long as their nerves are not compressed. (3 figures, 12 references)

Alice R. Deutsch.

Berinin, Goodwin M. **The nature of vergence revealed by electromyography.** A.M.A. Arch. Ophth. **54**:407-409, Sept., 1955.

Hering's and Sherrington's laws are corroborated by electromyography. (8 figures, 3 references) G. S. Tyner.

Dejean, C., De Jaeger, Sevrin, Danis, P., Hugonnier, Broun-Vallon, and François, J. **Discussion of R. Weekers' and Pierre Daenen's report on the surgical treatment of paralytic squint.** Bull. Soc. belge d'opht. **109**:1-15 Feb., 1955.

Dejean stresses the uncertainty in the prognosis as far as a permanent surgical restoration of function in certain types of paralytic squint is concerned. He quotes a personal observation of a young woman in whom he had repaired a paralytic convergent squint when the patient was 11 years old. For several years the surgical result was very good until the abducens paresis recurred at the age of 17 years, combined with signs of paralysis of other cranial nerves and of disturbance of the pyramidal tract. At this time the diagnosis of a tumor of the ponto-cerebellar angle was made. The original abducens paresis, which now was combined with a homolateral facial paralysis, apparently was the first sign of the growth so many years earlier.

De Jaeger emphasizes the fact that the

movements of the eyes after transplantation of muscles depends on the actions of the muscle and not just on the elasticity of the transplanted fibers and he provides some schematic sketches to clarify his concept.

Sevrin calls the folding of the superior oblique muscle temporal to the insertion of the superior rectus his method of choice. He had excellent results combining it with a marginal myectomy of the inferior oblique muscle.

P. Danis reemphasizes the importance of recognizing overaction of the contralateral synergist as a physiologic phenomenon in contradistinction to the overaction of the homolateral antagonist which is a pathologic occurrence. He stresses the need of an adequate evaluation of this fact before surgery. He also calls attention to the possible errors in differential diagnosis of paralytic and concomitant squint in children and reports a pertinent case history. Hugonnier uses the conjunctival approach to the inferior oblique if the weakening of this muscle must be combined with surgery of other muscles. For an isolated intervention on the inferior oblique muscle he prefers the subcutaneous approach. Braun-Vallon discusses the theoretical explanations of the possible results of muscle transplantation in paresis of the external rectus muscle. J. François presents an analysis of 200 cases of paralytic squint and a special report of an individual case. He also provides photomicrographs of an external rectus muscle in a case of Duane syndrome to demonstrate the lack of structural anomalies in this muscle. (2 figures)

Alice R. Deutsch.

Dubois-Poulsen, A., Beaumansour, and Attane. **Two cases of concomitant convergent squint, evident only in adulthood.** Bul. Soc. belge d'opht. **109**:60-74, Feb., 1955.

Concomitant strabismus is an affection of childhood and only rarely occurs in later years. Nevertheless concomitant squint has been observed in adults. Several characteristics have been described. It always develops in a short time and it proves to be very distressing to the patient. These facts gave the symptom complex its name —strabismus gravis. Neuropathic personality, physical or psychological trauma, minor eye injuries which necessitate patching of one eye seem to be predisposing factors. The angle of squint always is considerable and equal in all directions of gaze. It mostly occurs in myopes but also has been observed in others.

Two patients who suddenly acquired concomitant convergent squint in later life were studied by the authors. Many interesting facts were found while testing their visual fields. Zones of peripheral neutralization, displacements of targets, horizontal transposition and islands of normal correspondence were found when using polarizing filters. These findings confirmed the diagnosis of concomitant strabismus. Surgical treatment gave good results in one case.

The literature is reviewed and tentative explanations of the pathogenesis of this symptom-complex are offered. (4 figures, 16 references) Alice R. Deutsch.

Focosi. **Partial marginal myotomy in mild hyperfunction of the inferior oblique.** Bul. Soc. belge d'opht. **109**:85-88, Feb., 1955.

Partial marginal myotomy of the inferior oblique muscle was done in eight cases of esotropia and in two cases of exotropia in combination with mild overaction of the muscle. After exposure of the insertion of the muscle, 2 or 3 incisions were made in its inferior margin. The incisions were 2 to 3 mm. long and were made into the substance of the muscle itself and not into its tendon. The operation is recommended because of its simplicity and the rareness of overcorrection.

Alice R. Deutsch.

François, J. **Muscle transplants for the treatment of ocular palsies.** Bull. Soc. belge d'opht. **109**:88-105, Feb., 1955.

The operation discussed, consists of transplantation of the temporal halves of the superior and inferior rectus muscles to the stump of the resected external rectus. After exposure and section of the external rectus muscle a triple-armed suture is placed in its superior and inferior margins and afterward in the detached lateral half of the superior and inferior rectus musles, after a 1 cm. and 1.5 cm. longitudinal incision had been made in the upper and the lower muscle, respectively. The free ends of these sutures are placed in the stump of the external rectus muscle. After tying the three sutures carefully the transplanted muscle fibers slip under the shortened external rectus muscle. It is emphasized that the incision in the inferior rectus muscle must be longer than the one in the superior to avoid a possible post-surgical hypophoria.

The operation was successfully performed in 14 cases of congenital abducens paralysis, in four of which the paresis was bilateral. The average abduction achieved was 40°. A muscle transplantation was also tried in one case of congenital superior rectus muscle paralysis but was not successful. The author believes that the good surgical and functional results are to be ascribed chiefly to the elasticity of the tissue, and not to regeneration of nerve. (14 figures, 14 references)

Alice R. Deutsch.

François, J., and Derouck, A. **Electro-oculographic study in ocular palsies.** Bull. Soc. belge d'opht. **109**:23-49, Feb., 1955.

In this excellent paper the technique and the diagnostic values of electro-oculography are described and some details of the amazing results achieved are discussed. Four electrodes were used: electrode 1 was placed below the nasal border of the inferior orbital margin, electrode 2 at the temporal canthus, electrode 3 nasally on the superior orbital margin and electrode 4 halfway between the external canthus and ear. The horizontal movements were registered by leads 1-2 and 2-4, the vertical movements by leads 2-3 and 3-4. The potentials were magnified by an amplifier and registered on paper by an electromagnetic oscillograph on a time constant of 0.1. The ocular movements caused a deflection of the isoelectric line. The deflection is monophasic and the amplitude depends on the psychologic state of the person examined, the health and condition of the retina and the amplitude and frequency of the ocular movements.

Records of eight patients with various muscular anomalies are reviewed in detail. Characteristic modifications of the oculographic curves were seen in complete or incomplete external ophthalmoplegias, namely, absence of the deflection of the isoelectric line, irregular deflection, diminished amplitude and nystagmiform movements. In mild or healed ophthalmoplegias, abnormalities in the tracing only appeared in slight and fine movements as they occur during reading or testing of optokinetic nystagmus. In concomitant squint of paretic origin the oculographic curve may reveal the correctness of the diagnosis. (33 figures, 26 references)

Alice R. Deutsch.

Hambresin, L. **Ocular palsies and automobile accidents.** Bul. Soc. belge d'opht. **109**:49-53, Feb., 1955.

Among 250 records of compensation for accidental injury, mostly in automobile accidents, 27 persons were listed as having ocular palsy. The most frequent paralysis affected the oculomotor nerve either completely or only in its external division. The vulnerable position of the third cranial nerve at the petrosphenoidal ligament and at the external side of the pos-

terior clinoid process is emphasized. The possibility of hemorrhage into the nerve sheath simultaneously with a lesion of the superior petrosal sinus is described and the comparatively favorable prognosis of such an event is discussed. The high percentage of damage to the sympathetic (14.8 percent) was a surprising fact in this survey. Visual disturbances were comparatively rare. Among the 27 patients only four had impairment of vision. One patient had optic atrophy, two had homonymous hemianopsia and one a bitemporal hemianopsia. Alice R. Deutsch.

Jonkers, G. H. **D.F.P. in treatment of convergent squint. (A preliminary report)** Bull. Soc. belge d'opht. **109**:74-79, Feb., 1955.

D.F.P., in 0.025 or 0.05-percent solution was used in an unselected group of children with permanent or periodic convergent squint. Most of these children had been treated previously with occlusion, orthoptics and adequate optical correction. The therapeutic effect of D.F.P. was not especially favorable. However a certain diagnostic importance might be achieved with D.F.P. if a reliable relationship between the fixed and variable angle of squint could be recognized with this drug. (1 table, 3 references)

Alice R. Deutsch.

Kurus, Ernst. **External progressive ophthalmoplegia.** Klin. Monatsbl. f. Augenh. **127**:302-307, 1955.

Three typical cases are reported; in all three ptosis was manifest before other extraocular muscles became involved. The muscles could not be examined histologically, but a stellate ganglion block accentuated the ptosis in all three patients which suggests that neither the sympathetic nerve nor Müller's muscle is affected. A modified Friedenwald-Guyton sling operation is used to correct the ptosis. (3 figures, 10 references).

Frederick C. Blodi.

Ryan, H. **Divergent strabismus.** Australian & New Zealand J. Surg. **25**:154-156, 1955.

The author reviews 145 cases of divergent squint and classifies them into 1. tonic-constant angle, 2. small-angle divergence, and 3. divergence excess. For tonic squints orthoptics have little result, early operation is indicated. For small-angle divergence orthoptic control is advised. For divergence excess one quarter were treated by operation (recession of lateral rectus and resection of medial rectus) and three quarters were treated by orthoptics.

Ronald Lowe.

Schenk, H. **Experiments on the artificial paresis of extraocular muscles.** Klin. Monatsbl. f. Augenh. **127**:308-316, 1955.

Rhaetocain, a longlasting local anesthetic, is injected into the belly of one extraocular muscle. This causes a paresis of the muscle for several days with improvement of the angle of deviation in strabismus. In nine patients with exotropia the deviation decreased temporarily and about six months elapsed before the original condition was restored. The effect on esotropia was negligible. However, when the injection was done postoperatively three of seven patients showed a definite improvement. (3 references)

Frederick C. Blodi.

7

CONJUNCTIVA, CORNEA, SCLERA

Ambrosio, Andrea. **The repair of corneal wounds with lyophilized erythrocytes.** Arch. di ottal. **59**:333-343, July-Aug., 1955.

Eritrocilline, a combination of lyophilized human erythrocytes and 10,000 units

of penicillin per gramme, was used in the treatment of experimental corneal abrasions and cuts of rabbits. One eye was given a 5-percent solution of eritrocilline, the control eye a penicillin solution. The healing process was accelerated significantly when eritrocilline was used, and the resulting scars were smoother. On two patients with perforated corneal ulcer beneficial results were observed. (4 figures, 1 table, 43 references)

John J. Stern.

Cucco, Giovanni. **Subepithelial keratitis. (Clinical significance and various morphological aspects.)** Arch. di ottal. **59**:317-332, July-Aug., 1955.

Seven patients are described in whom minor trauma of the eye was followed after a few days by subepithelial opacities of various forms. The author regards this subepithelial keratitis as a post-traumatic viral infection. (25 references)

John J. Stern.

Cucco, Giovanni. **The corneal complications of epidemic keratoconjunctivitis.** Rassegna ital. d'ottal. **24**:166-182, May-June, 1955.

The author had an opportunity to study a large epidemic of the disease in the Naval Yards of Genoa. He states that keratoconjunctivitis is a definite disease entity but that symptoms and signs vary greatly. The corneal changes are the essential feature and were seen in 30 percent of the cases studied. The keratitis seen in the later stages are classic subepithelial virus changes. Three cases were selected and followed daily for some months wherein chronological notes confirm the belief that corneal lesions pass through several distinct phases. (30 references)

Eugene M. Blake.

DeLeonibus, Fernando. **Ophthalmometric examinations of a case of bilateral corneal ectasy.** Arch. di ottal. **59**:353-360, July-Aug., 1955.

A corneal ectasia which was neither a keratoconus nor a megalocornea was examined with the Helmholtz ophthalmometer. It was characterized by an irregular anterior surface with absence of the optical zone, by marked asymmetry and by curvature values highly divergent from the physiologic ones. (2 graphs, 2 tables, 5 references)

John J. Stern.

Friedman, B., Borrelli, F. J., and Geleris, I. **Lymphosarcoma of the bulbar conjunctiva.** AM.A. Arch. Ophth. **54**:381-385, Sept., 1955.

A case is reported in which a clinical cure was obtained by treatment with a radioactive strontium applicator. A total of 6600 beta r was applied with no apparent ill effect. (7 figures, 8 references)

G. S. Tyner.

Liebman, Sumner D. **An unusual case of filamentary keratitis.** A.M.A. Arch. Ophth. **54**:434-435, Sept., 1955.

A case of filamentary keratitis occurring in a squinting eye was cured by correction of the squint and establishing normal contact between the cornea and lid. (1 figure, 1 reference)

G. S. Tyner.

Zekman, T. N., and Krimmer, B. M. **The treatment of conical cornea.** A.M.A. Arch. Ophth. **54**:481-488, Oct., 1955.

A fluidless corneal lens with multiple inside curves designed for use in severe keratoconus is introduced. Studies carried on by the authors suggest that the fluidless lens is superior to the fluid lens in this disease because artificial fluids tend to increase corneal hydrops. The multicurve design of the new lens permits more accurate fitting and longer wearing time when the corneal apex is acutely curved. In some instances it was beneficial in reducing the apical corneal curve after use

of the lens. (6 figures, 9 tables, 14 references) G. S. Tyner.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Wilson, W. A., and Irvine, S. R. **Pathologic changes following disruption of blood supply to iris and ciliary body.** Tr. Am. Acad. Ophth. **59**:501-502, July-Aug., 1955.

The authors describe atrophy of the uveal tract which, they believe, may be ascribed to destruction of the major blood supply of the iris, ciliary body and choroid on one side in the course of retinal detachment surgery. Apparently the long posterior and the anterior ciliary arteries were injured. Microscopic preparations are described. Theodore M. Shapira.

9

GLAUCOMA AND OCULAR TENSION

Becker, B., and Constant, M. A. **Experimental tonography.** A.M.A. Arch. Ophth. **54**:321-329, Sept., 1955.

The alteration in rabbit aqueous flow induced by carbonic anhydrase inhibition was studied quantitatively. Systemic administration of acetazoleamide resulted in suppression of approximately 63 percent of aqueous secretion. This figure represents a maximum which cannot be exceeded by increasing the drug dosage, that is, 25 to 40 percent residual of normal aqueous flow cannot be inhibited regardless of the amount of drug given. Some eyes, although showing decreased aqueous formation, did not have a corresponding fall in intraocular pressure. These "pseudoresistant" eyes had a compensating decrease in aqueous outflow. (7 figures, 4 tables, 5 references) G. S. Tyner.

Berens, Conrad. **Glaucoma surgery.** A.M.A. Arch. Ophth. **54**:548-563, Oct., 1955.

The author believes cycloelectrolysis is more effective than cyclodiathermy in reducing aqueous formation. The long range outlook for eyes which have been so treated is not encouraging. Either procedure should be reserved for eyes in which filtering procedures have failed. (4 figures, 12 tables, 41 references) G. S. Tyner.

Dominguez, D., and Baranco, R. **Tonometric compression in the normal and in the glaucomatous eye.** Arch. Soc. oftal. hispano-am. **15**:483-487, May, 1955.

The author performed tonography, using the Schiøtz instead of the electronic tonometer, on 129 normal and 101 glaucomatous eyes. The data confirmed the increased resistance to outflow with age, a finding recorded first by Goldmann. A decided difference is seen in the data of the congestive and of the simple forms of glaucoma. Resistance was increased in both types of glaucoma, but the secretion of aqueous was found below normal in simple glaucoma, and above normal in congestive glaucoma. In two patients the authors found that the increased secretion of aqueous preceded the increased resistance to outflow. The authors also encountered cases in which the resistance to outflow was normal, the hypertension being caused entirely by hypersecretion of aqueous. Congestive glaucoma appears to set in with an overproduction of circulating aqueous, to which is added later an increased coefficient of resistance. As the glaucomatous process advances the coefficient of resistance increases, and secretion of aqueous diminishes, so that in absolute glaucoma the coefficient of resistance is very high, and secretion of aqueous low as a result of atrophy of the ciliary body. Simple glaucoma is characterized by an initial increase of resistance with small

quantities of aqueous; both are senile manifestations. The diminution in aqueous is the result of senile atrophy of the ciliary body. There are rare cases of glaucoma, diagnosed clinically as simple, which show an increased secretion of aqueous and but a slight increase of the coefficient of resistance. The authors consider these cases as compensated cases of congestive glaucoma. Ray K. Daily.

Foote, F. M., and Boyce, V. S. **Screening for glaucoma.** J. Chronic Dis. **2**:487-490, Oct., 1955.

In a number of surveys held in various parts of the country, the incidence of undiagnosed glaucoma in subjects aged 40 to 65 years, was found to be about two percent. In one group of 10,000 subjects there were 153 with glaucoma, 71 were reported as having border-line glaucoma, and 100 were kept under observation. When the Schiøtz tension was over 25 mm. Hg, visual field, gonioscopy, slitlamp examination, tonography, fundus examination and provocative tests were done. It was found that an ophthalmologist can screen 20 persons in an hour, with the help of an assistant who keeps the records and tests visual acuity. (9 references) Irwin E. Gaynon.

Koskenoja, M., and Esko, O. **Routine tonometry for glaucoma detection in geriatric patients.** Geriatrics **10**:362-365, Aug., 1955.

Tonometric examinations were done on 600 people over the age of 64 years; in 96 cases the ocular tension was 25 or over. There were 13 cases of known glaucoma, and 28 cases of primary glaucoma were found, giving an incidence of 4.6 percent in this age group. (1 figure, 1 table, 10 references) Irwin E. Gaynon.

Magdaleha Castineira, Jaime. **The compression test with the electronic tonometer.** Arch. Soc. oftal. hispano-am. **15**: 599-612, June, 1955.

After a review of the literature on the compression test the author proposes a new calculation for the determination of the rate of production of aqueous humor, based on the time required for the recovery of the initial ocular tension. Recovery curves were established from measurements on ten normal patients. There appears to be a physiologic increase of resistance to outflow with age, which explains the increased incidence of glaucoma with advancing age. In 32 eyes with chronic noninflammatory glaucoma the resistance to outflow was found increased, and the production of aqueous retarded. This is probably due to a self-regulating mechanism in the eye; when resistance to outflow was diminished, by surgery for example, the production of aqueous was restored to normal. In four cases of glaucoma without increased intraocular pressure resistance to outflow was always found increased. In some cases in which compression, even with a ten-gram weight, produced no fall in ocular tension, the angle of the anterior chamber was found, by gonioscopy, to be completely open, indicating that the cause of the increased ocular tension could not be an obstruction of the angle by the iris root. Even in such cases the instillation of pilocarpine for several days permitted carrying out of the compression test, although the resistance to outflow was very high. The author believes that this indicates that the increased resistance to outflow in such conditions is caused by an increased intravenous pressure. This fact may also explain the rare findings of an increase in intraocular pressure under compression. Among 36 cases of unilateral glaucoma increased resistance to outflow was found in 13 of the normal eyes. Referring to a statement recently published by Weekers, to the effect that no reliable case of increased intraocular pressure caused solely by an overproduc-

tion of aqueous has as yet been reported, the author reports a case of Meniere's disease in a woman, 34 years old, with an increased ocular tension, no increase of resistance to outflow, and a marked increase of production of aqueous. (2 graphs, 3 tables, 21 references) Ray K. Daily.

Spencer, R. W., Helmick, E. D., and Scheie, H. G. **Tonography.** A.M.A. Arch. Ophth. **54**:515-527, Oct., 1955.

In this article helpful suggestions are given to those beginning the use of tonography and pitfalls in the use of this complicated apparatus are outlined. It is important 1. to insure free venous flow from the head to neck of the patient (no constricting collar or necktie), 2. that the patient maintain fixation, 3. to retract the lids adequately (the authors suggest the use of a non-magnetic speculum), 4. to hold the tonometer steady in a vertical position, 5. to calibrate the tonometer with the recording device (electric tonometers require a special resistor jack and a constant voltage transformer to give an accurate tracing), and 6. to properly care for the tonometer. Ether cannot be used because it dissolves insulation. It should be cleaned with alcohol, wiped with distilled water and dried with a lintless cloth. Control studies indicate that there is an error of 0.04 in tracings of all eyes. Once the tonometer is properly calibrated, accurate determinations of facility of aqueous flow can be made without the use of the recorder. (7 figures, 7 tables, 15 references) G. S. Tyner.

Sysi, R. **Effect of "Myanesin" on intraocular pressure.** Brit. J. Ophth. **39**: 619-622, Oct., 1955.

This drug is suggested as a substitute for curare since it definitely relaxes muscles yet does not give the undesirable side effects. It is administered intravenously and produces some vertigo and sensation of heat in the skin. No other side effects were noted. A definite fall in pressure was noted almost immediately, particularly in the acute shallow-angle and in the secondary type of glaucoma. This drop tends to disappear within 20 to 25 minutes after the injection, but the tension can be made normal by the use of miotics. (2 figures, 5 references) Lawrence L. Garner.

Weekers, R., and Watillon, M. **The treatment of glaucoma by reducing aqueous humor formation.** Ann. d'ocul. **188**: 654-664, July, 1955.

Systemic use of diamox, topical application of adrenalin, and retrociliary diathermy are three methods by which the formation of aqueous humor may be diminished. Each probably acts through a different process.

In narrow-angle glaucoma, diamox is used in association with miotics in the acute attack. However, if miotics alone do not control the tension, surgery is indicated. Adrenaline is contraindicated, as it may produce mydriasis even when used with D.F.P.

In open-angle glaucoma diamox can be used over long periods of time; 0.01-percent D.F.P. and 2 percent adrenaline drops instilled once a day, either separately or together, are often effective in early cases. In more advanced cases, iridencleisis ab externo is the treatment of choice. Retrociliary diathermy is an important adjunct if efforts to improve aqueous outflow are not successful.

In glaucoma complicating active uveitis adrenalin, diamox, and retrociliary diathermy are of value in those few cases in which the abnormality is not controlled by atropine and cortisone. In glaucoma following uveitis adrenalin and retrociliary diathermy are still of value, and diamox is less so. In the more resistant cases iridencleisis may be required.

In glaucoma due to thrombosis of the central vein, diamox is of little value. The response to iridencleisis is also poor. Re-

peated diathermies are sometimes required, and are often successful only after almost all activity of the ciliary body has been abolished. (4 tables, 15 references) John C. Locke.

10

CRYSTALLINE LENS

Avasthy, P. **Myanesin elixir in 150 cases of cataract extraction.** Brit. J. Ophth. **39**: 623-625, Oct., 1955.

Orally this new muscle relaxant was used in 2 dram doses one hour before operation in a series of cataract extractions in Kanpur, India. 200 patients selected at random were given no muscle relaxant and served as controls while "myanesin" was administered to 150 patients. Most of the entire group were of nervous temperament, uncooperative, uneducated and disturbed by a language difficulty. Tensions taken after the use of this drug showed only minimal reduction in a small number of patients, but there was a decided reduction in ocular motility and usually a relaxed patient. Vitreous loss occurred in 9 percent of the patients in the control group and in 1.3 percent of those who had been given myanesin. Myanesin seems to compare favorably with curare and has the advantage of a simple and economical method of administration. (2 tables, 7 references)

Lawrence L. Garner.

Barraquer Monar, Joaquin. **Corneoconjunctival flap sutures in cataract surgery.** Ann. d'ocul. **188**:572-576, June, 1955.

The author employs very fine silk on 4 mm. Grieshaber needles. He uses corneoscleral or scleroscleral sutures, which he buries completely under a limbus-based conjunctival flap. The latter is secured by plasma-thrombin solution. The sutures cause no irritation and are not removed. He uses the same fine silk and needles for keratoplasty. In the case of a 6-mm. perforating transplant, up to 16 such sutures can be placed, assuring minimal trauma and perfect coaptation. (6 figures)

John C. Locke.

Recupero, C. **The inclusion of the Ridley lens after extracapsular cataract extraction. (Personal cases)** Arch. di ottal. **59**:253-284, July-Aug., 1955.

In 15 patients a Ridley lens was implanted after extracapsular cataract extraction. In five cases complications were observed which necessitated removal of the implant. In other cases the postoperative irritation could be controlled with cortisone. The results—after a period of observation from 3 to 20 weeks—were satisfactory, even in those patients in whom the implant had to be removed. (1 table, 24 references) John J. Stern.

Silvan, F. **A new mechanically controlled erisophake for cataract extraction.** Arch. soc. oftal. hispano-am. **15**:659-661, June, 1955.

This is a report of a further modification in the design of the suction apparatus reported by the writer at the International Congress of Ophthalmology in New York. The objective of this erisophake is to free the hands of the surgeon of the task of maintaining compression of the rubber tip, required by the manually controlled erisophake. In the author's design the rubber tip is enclosed in a casing, which contains in addition a device for compressing the rubber tip, controled by a pedal through a cable. (2 figures)

Ray K. Daily.

11

RETINA AND VITREOUS

Bellavia, Marco. **Jensen's chorioretinitis (clinical case).** Arch. di ottal. **59**:377-391, Sept.-Oct., 1955.

On the basis of a clinical observation and a review of the literature the author discusses the etiology and recommends that the descriptive term juxtapapillary be dropped in favor of chorioretinitis of Jensen. (3 figures, 31 references)

John J. Stern.

Bennett, George. **Central serous retinopathy.** Brit. J. Ophth. **39**:605-618, Oct., 1955.

The author analyses his findings in 27 cases of serous retinopathy against a background of modern conception of this disease. Relationship to disciform degeneration of the macula is noted: the latter usually occurs in older subjects and the former between the ages of 25 to 45 years. Males are most frequently affected and the visual prognosis deteriorates significantly as the age rises. The disturbance is most commonly bilateral and often occurs in individuals with a nervous or tense or emotional instability. Medical treatment is discussed and psychotherapy is suggested. (8 tables, 111 references)

Lawrence L. Garner.

Manschot, W. A. **Therapy of retrolental fibroplasia.** A.M.A. Arch. Ophth. **54**:596-601, Oct., 1955.

The necessity for accurate control of oxygen concentrations and withdrawal of oxygen is demonstrated. A concentration of 40 percent is considered optimal. If signs of retinal edema recur after withdrawal, oxygen should again be administered. (2 figures, 20 references)

G. S. Tyner.

Marchessi, F. **A case of retinal tear without detachment followed for two years. A discussion of the pathogenesis of retinal detachment.** Arch. soc. oftal. hispano-am. **15**:662-681, June, 1955.

The patient, 50 years old, was a myope of seven diopters in each eye. In the course of three years, from 1948 to 1951, he had five operations for recurrent retinal detachment of the right eye. Three tears were closed by diathermy coagulation combined with air injection into the vitreous. Vision of 5/10 was maintained since the last operation. In June, 1952, the patient became disturbed by cloudy vision in the left eye and was found to have in that eye a turbid vitreous, and a tear in the 11-o'clock meridian, with a floating operculum, the base of which was toward the ora serrata. With rest and stenopeic spectacles the hemorrhage was absorbed. In the course of two years the patient had three other hemorrhages, which subsided with the use of stenopeic glasses, and the tear remained without producing a detachment. To explain this different behavior of the two eyes of the same patient, the literature on the pathogenesis of retinal detachment is reviewed, and the following conclusions reached: the important factor in retinal detachment is a secretory disturbance of the pigment epithelium; the subretinal fluid consists chiefly of the secretion of irritated pigment epithelium, joined later by degenerated vitreous. A retinal tear including the external limiting membrane is the provocative factor for the secretory disturbance, and subsequently the movement of the detached vitreous upsets the secretion-absorbtion process of the epithelium. If the retinal tear does not include the external limiting membrane the retina does not become detached. Therefore the author divides idiopathic retinal detachments into three groups: 1. detachments with a complete tear, 2. detachments without a tear but in which a tear in the detached retina may occur later; and 3. incomplete tears, which do not cause retinal detachment as long as the external limiting membrane remains intact. The author's case falls within the last category. (23 references) Ray K. Daily.

Morone, Giulio. **The role of the mucopolysaccharides in diabetic retinopathy.** Rassegna ital. d'ottal. **24**:161-165, May-June, 1955.

The author studied the behavior of the glucosamines of the vitreous mucoids in diverse stages of experimental diabetes. The aim was to draw some inferences concerning the role played by the mucopolysaccharides. There was evident a parallelism between vascular modifications and the behavior of the polysaccharides in question. It did not appear unreasonable to the experimenter to conclude a probable intervention of the metabolism of the mucosaccharides and the vascular complications of diabetes. (2 figures, 17 references) Eugene M. Blake.

Palich-Szanto, O. **Late sequel of a thrombosis of the central retinal vein.** Klin. Monatsbl. f. Augenh. **127**:358-364, 1955.

A disc-shaped elevation was noted 18 months after occlusion of the central vein beneath the macula. It was somewhat larger than the papilla and had a central depression. Vessels ended in it and it probably was an organized hemorrhage. (1 figure, 36 references)

Frederick C. Blodi.

Rusodimos, C. N. **Central angiospastic retinopathy.** Arch. Soc. oftal. hispano-am. **15**:530-538, May, 1955.

Seven cases are reported, five in women and two in men. The age of the patients ranged from 39 to 50 years. All patients had neurotic manifestations. Only one smoked. Five patients recovered under treatment. One recovered spontaneously. One case ended with degeneration of the macula. Of the therapeutic agents antibiotics and cortisone proved to be ineffective. Sedatives, vasodilators, and vitamins B, D, and E constituted the other medicaments used. (6 references)

Ray K. Daily.

de Vincentiis, Mario. **The pathogenesis of the macular hole.** Arch. di ottal. **59**: 345-352, July-Aug., 1955.

A statistical survey of 57 cases with or without retinal detachment indicates the possibility that a hole in the macula can be secondary to the detachment, particularly when peripheral retinal lesions are present. (4 tables, 8 references)

John J. Stern.

de Vincentiis, Mario. **Observations on retinal detachments in myopic and nonmyopic eyes.** Ophthalmologica **129**:353-369, June, 1955.

The author has made a search for systematic differences between the retinal detachments that occur in myopes and those that occur in nonmyopes. For the purpose of this study an eye was considered myopic if a myopia of seven or more diopters was present, and as nonmyopic if its refraction was emmetropia or hyperopia. Fifty cases of detachment of each type were collected and studied. A history of injury was elicited with significantly greater frequency in the nonmyopes. In the latter type of eye the detachment tended to occur later in life than in the myopes. A significant difference between the two groups was found with regard to the location of the retinal breaks. In the myopes the temporal half of the retina seemed to be predisposed to break formation whereas in the nonmyopes the distribution of the breaks was more even. From this observation the author draws the conclusion that in the myopes the extrinsic ocular muscles, by traction or pressure, play an important role in the causation of retinal breaks. In the nonmyopes the action of the extrinsic muscles is partly compensated by the soundness of the ocular tissues and partly masked by the effects of trauma. (2 figures, 17 tables, 16 references) Peter C. Kronfeld.

Weerekoon, Lloyd M. **Priscol and reti-**

nal artery occlusions—preliminary report. Brit. J. Ophth. **39**:98-102, Feb., 1955.

Three cases of retinal artery occlusion are described in which therapy was given within a few hours in the first two cases and after seven days in the third. The results from retrobulbar injection of Priscol coupled with amyl nitrite inhalation suggests strongly that this method be used with other known forms of vasodilatation. The author advises that intensive vasodilatation should not be withheld even in what may appear to be a hopeless case, and that therapy should continue for at least thirty days. The retrobulbar dose of Priscol varies from 0.01 g. to 0.025 g. as the initial treatment to be followed by the oral use of Priscol at regular intervals. Toxic effects have been described in the literature, but the author noted only flushing of the face and feels that his dosage was too low to produce toxic effects. Pain and ocular palsies which are sometimes seen after the retrobulbar injection of acetyl choline, were not noted here. (2 figures, 13 references)

Lawrence L. Garner.

12

OPTIC NERVE AND CHIASM

Landolt, E., **Optic nerve lesions in trauma of the skull.** Acta Neurochir. **4**: 128-142, Feb., 1955.

The author presents nineteen cases of unilateral optic lesions following trauma to the skull. Fractures of the lesser wing of the phenoid were found in three-fourths of the cases. The author presumes that in each case a fracture occurs, causing pressure on the optic nerve from edema or hemorrhage, rather than bone spicules, and advises early decompression to avoid the high incidence of permanent blindness which results. In all cases the injury was to the front half of the skull, and the most common finding was impaired pupillary function, which comes on very quickly. (3 figures, 19 references)

Harry Horwich.

Levine, S., and Bronstein, M. **The optic nerve sheath pathway.** A.M.A. Arch. Ophth. **54**:369-372, Sept., 1955.

Three cases are presented which provide evidence for the transmission of particulate matter through the optic nerve sheath from the cranial cavity to the orbit. In one case a foreign body granuloma of the sclera resulted after intracranial surgery. In two other cases there was infiltration with tumor cells. (5 figures, 9 references) G. S. Tyner.

Love, J. G., Dodge, H. W., Jr., and Blair, H. L. **Complete removal of gliomas affecting the optic nerve.** A.M.A. Arch. Ophth. **54**:386-391, Sept., 1955.

Four cases are reported in which complete removal of the tumor was accomplished through the transcranial approach. (7 figures, 12 references) G. S. Tyner.

Marchessi, Fernando. **Late bilateral hereditary optic atrophy.** Arch. Soc. oftal. hispano-am. **15**:522-529, May, 1955.

This is a report of a case of Leber's optic atrophy in a man 59 years old. The genetics of this affection are reviewed, and the differential diagnosis discussed. (Genealogic tree) Ray K. Daily.

Nonnenmacher, Heinz. **Optic neuritis in lactation.** Klin. Monatsbl. f. Augenh. **127**:228-233, 1955.

A 35-year-old woman noted a sudden loss of vision in the left eye one month after her second delivery. There was a papillitis in the left eye and amaurosis. Three months later light perception was present. There was a slight temporary retrobulbar neuritis on the other side. The author cautions against the diagnosis of a lactation neuritis especially as he saw a similar case where the woman developed

multiple sclerosis afterward. (16 references) Frederick C. Blodi.

Oehninger, C., Barrios, R. R., and Haedo, C. A. **Optic neuritis caused by arsenicals. Treatment with B. A. L.** Brit. J. Ophth. **39**:422-428, July, 1955.

Two patients who had had stovarsol for syphilis noted cloudy vision after several weeks. Central vision was good but there was marked constriction of peripheral fields. The optic discs were slightly pale and neither patient improved until given injections of B. A. L. intramuscularly in amounts of 200 to 300 mg. daily. Both patients improved. (6 figures, 13 references) Morris Kaplan.

13

NEURO-OPHTHALMOLOGY

Adler, E., Landau, J., and Rabinowitz, E. **Palsy of conjugate eye movements in poliomyelitis.** Conf. Neurologica **15**:154-161, 1955.

The overall incidence of ocular disturbances in bulbar poliomyelitis is about 10 percent. Nystagmus is the commonest finding, being possibly a residuum of transient paresis. Next in order come pareses of the abducens, oculomotor, and trochlear nerves, and least commonly, pupillary disturbances. Reporting on the Israeli epidemic of 1949-54, they noted four cases of paresis of conjugate gaze, and suggest that the apparent rarity of this finding may be due to the transient nature of the palsy. They conclude that it is due to a lesion in the pontine center, possibly the vestibular nucleus. (3 figures, 14 references) Harry Horwich.

Agarwal, Lalit P. **Vascular headaches.** Ophthalmologica **129**:370-376, June, 1955.

Under the heading of vascular headaches the author considers those which occur in patients with periodic headaches, unilateral or generalized in onset, associated with a wide variety of other symptoms (nausea, lacrimation, photophobia, mental depression or anxiety) and often announced by visual prodromes. For the treatment in these cases the author has used dihydroergotamine combined with pyrazolon derivatives (irgapyrin) with considerable success. (1 table, 29 references) Peter C. Kronfeld.

Albert, P., and Aznarez, J. **Foster-Kennedy syndrome without a tumor. Edematous intracanalicular optic neuritis.** Arch. Soc. oftal. hispano-am. **15**:509-516, May, 1955.

The author reports a case of bilateral intracanalicular optic neuritis, clinically suggestive of an intracranial lesion, as the patient had an optic atrophy in the right eye, and an optic neuritis in the left. Encephalography with air injection was negative for tumor. The patient recovered normal visual fields and visual acuity in the course of several months under treatment with Vitamin B_1. The only remaining sequel of the disease was the optic atrophy of the right disc. The author concludes that the Foster Kennedy syndrome is not pathognomonic of an intracranial lesion, and the possibility of an intracanalicular optic neuritis should be kept in mind in its presence. (2 figures, 3 references) Ray K. Daily.

Ellerbrook, V. J. **Unilateral Bell's palsy—a case report.** Am. J. Optometry **32**:579-582, Nov., 1955.

A school teacher, aged 37 years, was fitted successfully for a contact lens over an eye which would not blink enough to prevent drying of the cornea. A forced closure of the eye resulted in an ugly grimace, so she closed the eye with her finger every few minutes.

Paul W. Miles.

François, J., and Verriest, G. **Perimetry of the dark-adapted eye in neuroöphthalmology.** Ann. d'ocul. **188**:589-653, July, 1955.

The authors discuss in detail the comparable effects on photopic and scotopic visual function of various neuroöphthalmic lesions. A determination of the size of the central scotoma of the dark-adapted eye constitutes a new method of clinical examination of considerable practical interest, since it allows the detection of changes too small to be perceptible by methods of photopic examination. (38 figures, 58 references) John C. Locke.

Gormaz, Alberto B. **Neuro-ophthalmologic disturbance in barbiturate poisoning.** Arch. chil. de oftal. **12**:82-84, Jan.-June, 1955.

The author describes a patient, convalescing from a barbiturate poisoning, who had mydriasis, paralysis of accommodation, paresis of the medial rectus muscle of the left eye, papilledema and a homonymous defect in the upper right quadrant of the visual fields. All these symptoms cleared in a few weeks. (2 figures, 4 references) Walter Mayer.

Guibor, G. P. **Cerebral palsy: a practical routine for discerning oculomotor defects in cerebral palsied children.** J. Pediat. **47**: 333-339, Sept., 1955.

The importance of early diagnosis and treatment of the ocular manifestations of cerebral palsy is stressed. It is shown that not only the eyes, but also the whole organism will respond better. In addition to preserving vision and maintaining binocular function, better co-ordination of the arms and legs can be expected. Over fifty percent of cerebral palsy victims have ocular defects. It is demonstrated how these can be picked up by the Hirschberg corneal reflex test, versional tests for conjugate movements, and the testing of visual acuity with the illiterate "E" chart. (5 figures, 10 references) Harry Horwich.

Heinzen, H. and Baasch, E. **Ophthalmoplegia in congenital myasthenia gravis.** Ophthalmologica **129**:335-341, April-May, 1955.

To the five reported cases of congenital myasthenia gravis the authors add another. The patient, a now 15-year-old boy, was born of normal parents. Difficulty in swallowing was noticed shortly after birth. He learned to walk, but rapidly increasing fatigue and insecurity kept him from ever walking farther than a few blocks. All skeletal muscles were present, but small in volume. There was almost total bilateral external ophthalmoplegia with marked ptosis and normal visual acuity.

All muscular functions except the motility of the eyeballs improved dramatically and lastingly on oral administration of a prostigmine preparation. A biopsy from the biceps and from a rectus muscle revealed no histologic anomaly. (2 figures, 15 references) Peter C. Kronfeld.

Mancall, Irwin T. **Opticociliary neuritis.** A.M.A. Arch. Ophth. **54**:436-437, Sept., 1955.

The fifth case of optic neuritis associated with paralytic mydriasis and paralysis of accommodation is reported. (4 references) G. S. Tyner.

Montes, Galvez J. **The ocular symptomatology of tuberculous meninigis.** Arch. Soc. oftal. hispano-am. **15**:539-547, May, 1955.

Because of the new therapeutic agents, the prognosis of tuberculous meningitis has undergone a marked change. An affection which was fatal a few years ago, has been converted into a curable disease in a number of cases. The recovery, which

in such cases is slow and complicated by recurrences, permits careful study of the course and evolution of the ocular complications. This analysis of the ocular symptoms observed in 32 cases, is presented in a tabulated report. The most frequent pupillary disturbance was a tendency to mydriasis with a reduced amplitude of the photomotor reflex. Of the motor disturbances the most frequent was an involvement of the fourth pair of cranial nerves; the motor disturbances usually improved in the course of therapy. Tubercles of the choroid were found in 22 percent of cases, and their incidence was much higher in adolescents than in infants. The author points out that the presence of a tubercle of the choroid in a case of meningitis of undetermined etiology is diagnostic of tuberculosis. (2 tables) Ray K. Daily.

Nover, Arno. **Retinal changes in Schilder's disease.** Klin. Monatsbl. f. Augenh. **127**:294-302, 1955.

An eight and one-half-month-old child died after a short illness with the signs of a disseminated encephalitis. There were nystagmus, sixth nerve palsy and numerous white foci in the superficial layers of the retina. The histologic examination revealed many cellular deposits on the internal limiting membrane. There were in addition a slight edema of the retina and some perivascular changes. (3 figures, 62 references) Frederick C. Blodi.

Pallis, C. A. **Impaired identification of faces and places with agnosia for colours.** J. Neurol., Neurosurg. & Psychiat. **18**:218-224, Aug., 1955.

A unique case is described, in a patient with mitral stenosis and auricular fibrillation who had an embolus of the right posterior cerebral artery, as shown by angiography. This resulted in a bilateral superior homonymous quadrantanopia, color-blindness, inability to recognize faces, and disruption of spatial thought. Except for variations of the latter two, all his other functions were normal. Examples are given, wherein he did not recognize his wife or doctor, but could recognize animals, and so on. A detailed discussion of the case follows. (19 references) Harry Horwich.

Poleff, L. **Anisocoria of sympathetic origin in tuberculosis.** Ann. d'ocul. **188**: 665-671, July, 1955.

In a study of 301 patients with pulmonary, surgical, glandular and cutaneous forms of tuberculosis, anisocoria was found in 74.8 percent after a weak solution of atropine had been instilled and in 9.6 percent when atropine had not been instilled. The pupillary inequality was most common in pulmonary affections (100 percent) and least common in the surgical forms (58 percent). In pulmonary tuberculosis, the affected pupil was always on the side of the lesion. In early cases, it was larger than normal, and due to sympathetic irritation; whereas in cases of longer standing, there was miosis, due either to sympathetic paralysis or to a vagotonic reaction. When the location of the lesion was not related to the sympathetic nerve, the origin of the anisocoria remains unexplained. (21 references) John C. Locke.

Riesco MacClure, J. S. **Nystagmus. Its clinical significance.** Arch. chil. de oftal. **12**:7-32, Jan.-June, 1955.

In the study of 94 patients with cerebral lesions, the relation between the otoneurological signs and the cerebral area which actually presents the disturbance, the author reaches the conclusion that none of the following signs—spontaneous nystagmus in one or more directions, conjugate movement of the eyes, positional nystagmus, vestibular hyperexcitability, vestibular excitability with normal coclear

excitability, or absence of the rapid component of nystagmus—had any localizing significance. However, he believes that some other signs suggest strongly a lesion in the midline of the posterior fossa, but all of these signs should be evaluated with the neurological, otological, ophthalmological, and radiographic findings. (5 figures, 9 tables, 21 references)

Walter Mayer.

Venturi, G. **Total bilateral ophthalmoplegia in a case of postparotitic polyradiculitis.** Ann. di ottal. e clin. ocul. **81**:249-358, Aug., 1955.

In an eight-year-old child epidemic parotitis was followed by the syndrome of total bilateral ophthalmoplegia, meningeal irritation, motor deficiency in the limbs, progressing to a flaccid tetraplegia, and peripheral facial paralysis. The syndrome is interpreted as a polyradiculitis. Ten months after the onset there was still a disturbance of the ocular mobility, mydriasis and poor accommodation, while the tetraplegia had resolved into mild mobility disturbances of the limbs. (24 references)

John J. Stern.

14

EYEBALL, ORBIT, SINUSES

Arruga, H. **Problem of orbital implants after enucleation.** Brit. J. Ophth. **39**:599-604, Oct., 1955.

The inadequacy of implants is attested to by this famous surgeon's presentation of yet another type of implant. The implant differs from present modifications only in that it presents one or two noncorrodible metal pegs which protrude 7 mm. from the anterior surface. These are located at the upper portion of this anterior surface in order to keep the wound intact since perforation takes place at some distance from the wound which is sutured in the usual manner. The author's implant is acrylic and employs two sets of tunnels through which the vertical and horizontal rectus muscles are passed and then sutured in the usual manner. The purpose of the two pegs is to prevent rotation and lend stability. Conjunctiva completely covers the implant and the two pegs slowly perforate the overlying tissues in about two to four weeks. A semifinal acrylic prosthesis can be fitted in 60 to 90 days. A second type of procedure is described and is designed to avoid any buried sutures and no tunnels are required. The eye is prepared as in an evisceration, the scleral ring is made by cutting away all sclera at a point 10 mm. from the limbus. The implant is fitted into the scleral ring from behind by passing through the area between the inferior and lateral rectus muscles while small holes are made for the pegs in the sclera near the limbus. The latter procedure has been used only in five cases and is too new to evaluate fairly. The first procedure described has been employed by this author since 1951 in 39 cases and in five reoperation became necessary (19 figures)

Lawrence L. Garner.

Bossi, R., and Pisani, C. **Collateral cerebral circulation through the ophthalmic artery and its efficiency in internal carotid occlusion.** Brit. J. Radiol. **28**:462-469, Sept., 1955.

Three cases of ophthalmic collateral circulation, demonstrated by angiograms, are presented. The collateral circulation extended from the external carotid to the internal maxillary artery to the deep anterior temporal artery and thence to the zygomatic and temporal branches of the lachrymal artery. (13 figures, 31 references)

Irwin E. Gaynon.

Cutler, W. M. **Ocular manifestations of lethal midline granuloma: two cases.**

Univ. Michigan Med. Bull. **21**:220-228, July, 1955.

Two cases of Wegener's granulomatosis are presented, with the differential diagnosis and management of this invariably fatal disease. In these cases, steroid therapy was found to be helpful, but did not influence the final outcome. The case reports illustrate the various ocular complications which arise as the tissue invades the orbit. In both cases, a primary type of optic atrophy supervened. (2 figures, 9 references) Harry Horwich.

Foster, John. **Diagnosis and treatment of orbital tumors.** Roy. Coll. Ann. **17**:114-129, Aug., 1955.

Angioma is the commonest orbital tumor. Seventy percent of the tumors arise within the orbit, 23 percent from nearby structures, and seven percent from distant sources. The classical symptoms are exophthalmus, edema of the lids, defective vision with or without optic nerve changes, and diplopia. X-ray studies in 42 percent of cases, show increased tissue density, distention of the orbit, erosion, rarefaction, fossa or dent, or dilatation of the optic canal or sphenoidal fissure. A lateral orbitotomy rather than a transfrontal approach is advocated. (6 figures, 2 tables, 22 references)

Irwin E. Gaynon.

Laurent, L. P. E., Scopes, J. W. **Hyaluronidase in the treatment of exophthalmic ophthalmoplegia.** Lancet **2**:537-538, Sept. 10, 1955.

This 53-year-old man developed exophthalmic ophthalmoplegia following the cessation of thyrotoxic symptoms in the course of treatment with methyl thiouracil. He developed severe conjunctival edema with paralysis of the elevators of the eye and of the left external rectus muscle. 1,000 Benger units of hyalase dissolved in 0.4 ml. of 1-percent procaine was injected into the conjunctiva at four different times. The improvement brought to light the fact that the conjunctival edema was not in the fornix, but over the lower part of the globe. After retrobulbar injections, there was an improvement in the exophthalmus and the diplopia had ceased. (4 figures, 4 references)

Irwin E. Gaynon.

Naffziger, H. C.: **Progressive exophthalmos.** Bull. Am. Coll. Surgeons **40**:33-39, and 53-55, Jan.-Feb., 1955.

The condition, which appears usually after surgical removal of the thyroid gland but can occasionally be encountered in the absence of any detectable endocrine disturbance, is of a variable severity, ranging from a relatively mild degree to a few instances where an urgent intervention is required in order to forestall blindness and death from meningitis or brain abscess.

A marked, gradually increasing proptosis is soon followed by puffiness of the lids, epiphora, an edematous protrusion of the conjunctiva, limitation of ocular movements, corneal ulceration, choked discs and changes in the visual fields; most commonly visual impairment is associated with no changes other than the high retrobulbar pressure upon the optic nerves. This increased pressure has far more diagnostic and prognostic significance than the exophthalmometer readings, a fact which warrants the use of an orbitotonometer, especially in apparently unilateral cases. The etiology is still unsettled. From the pathologic standpoint, augmented fluid in the orbit seems to be the main cause for proptosis, while the orbital fat has been found almost absent; the extraocular muscles are swollen, infiltrated and may show loss of striation, fibrosis and even hyaline changes.

Hormonal treatment is of little avail, as is also X-ray therapy to the pituitary

or the orbital tissues. Surgical decompression of the orbit through a frontal approach has given, on the other hand, gratifying results in the 40 successive cases reported. No mention is made of Blaskovics' recession of the levator muscle, a procedure which is claimed by many to have been satisfactorily employed in cases of a not extreme gravity. (38 key references)

A. Urrets-Zavalia, Jr.

Pettinati, Sergio. **An unusual case of traumatic lesions of the orbit.** Rassegna ital. d'ottal. **24**:233-238, May-June, 1955.

Five years before the examination by the author a young farmer fell from a bicycle. A sharp stick penetrated the upper portion of the orbit. The stick was removed and after a few months exophthalmos developed and increased, accompanied by lagophthalmos. The eyeball was uninjured. X-ray study and exploratory surgery revealed an inflammatory granuloma which was removed. (4 figures)

Eugene M. Blake.

Senâ, Jose Alberto. **Intermittent exophthalmos.** Arch. oftal. Buenos Aires **30**: 133-139, April, 1955.

Arising almost invariably as the most striking symptom of orbital varices, this rare, essentially transient phenomenon appears sporadically whenever a cephalic venous stasis is induced either by rotating or tilting the head or by manual or instrumental compression of the jugular veins.

The case of a 24-year-old man is reported, in whom a marked intermittent proptosis of the left eye had been noticed when stooping or after exertion in the past four years. The condition was accompanied by only slight discomfort. On examination, a moderate left enophthalmos was seen (exophthalmometer readings were 16 mm. on the right side and 14 mm. on the left), which, by merely bending the head forward was replaced by a protrusion of up to 20 mm.; this subsided as soon as the head went back to an erect position. The eyeballs themselves were normal including the fundi, and vision was 20/20 in each eye. Apparently no bruit was heard, nor were there subjective murmurs or roaring perceived. Roentgenograms were not taken. As the case did not seem to be a clearly progressive one, all treatment was deferred. (5 figures, 8 references)

A. Urrets-Zavalia, Jr.

de Voe, Arthur Gerard. **The orbit.** A.M.A.. Arch. Ophth. **54**:438-461, Sept., 1955.

The pertinent literature for 1954 is reviewed. (328 references)

G. S. Tyner.

15

EYELIDS, LACRIMAL APPARATUS

De Conciliis, U. **A rare malformation of the lacrimal pathways.** Arch. di ottal. **59**: 401-408, Sept.-Oct., 1955.

A duplication of the lower lacrimal punctum and canaliculus is described. Both canaliculi communicated with the lacrimal sac. The upper punctum and canaliculus were absent. The author considers the condition an ectopy of the superior canaliculus. (4 figures, 39 references)

John J. Stern.

Debrousse. **The utilization of steel wire in oculopalpebral surgery.** Arch. d'opht. **15**:487-500, 1955.

The author stresses the value of steel wire in the repair of lid wounds, particularly in the initial repair. He deplores the fact that in some 70 percent of cases lid repair is accomplished by the nearest general surgeon and that the ophthalmologist is usually called in to correct the late unhappy results. He then discusses in detail the techniques of oculopalpebral

surgery and illustrates them with photographs and case reports. (6 figures)
P. Thygeson.

Dejean, Ch. **Causes of failure in lacrimal intubation.** Arch. d'opht. **15**:474-478, 1955.

The author refers to his initial report in 1953 of a study of 30 cases of lacrimal intubation, and states that advances in technique have now given him almost uniform success in several hundreds of cases. He notes that many ophthalmologists complain of lack of success and attempts in his article to analyze the causes of failure. Chief among these he believes to be the use of too small a tube. He considers that acrylic tubes of at least 2 mm. internal diameter are necessary, and he is now using tubes with internal diameter of 2.5 mm., a length of 15 mm., and with the lower end cone-shaped to facilitate its introduction into the canaliculus. He finds it necessary to enlarge the nasolacrimal canal by curetting the soft parts and employing a round file for the bony parts. It is important, he believes, to allow enough time to elapse after an acute dacryocystitis to prevent an exacerbation. Among factors of importance to be considered are anomalies of the nasal fossae and osteoperiostitis of the ethmoid. He concludes his paper with a detailed description of his technique. P. Thygeson.

Demorest, B. H., and Milder, B. **Dacryocystography.** A.M.A. Arch. Ophth. **54**: 410-421, Sept., 1955.

This is a sequel to a previous article by the authors and deals with demonstration of some of the commoner pathologic conditions encountered in dacryocystography. The authors believe that this procedure has a wide application. Among the conditions which can be demonstrated are functional block, anatomic block, atony or stenosis of the sac, fistula and diverticula, and the condition of surrounding structures which may influence therapy. (13 figures, 3 references) G. S. Tyner.

Forrest, Arnold W. **Epithelial lacrimal gland tumors. Pathology as a guide to prognosis.** Tr. Am. Acad. Ophth. **58**:848-866, Nov.-Dec., 1954.

The author discusses the management of patients on the basis of the pathologic diagnosis. He discusses briefly the confusion in the grouping of "mixed tumors" and describes histologically the findings in benign and malignant tumors. (28 figures, 1 table, 8 references)
Theodore M. Shapira.

Hughes, Wendell L. **Surgical treatment of congenital palpebral phimosis.** A.M.A. Arch. Ophth. **54**:586-590, Oct., 1955.

A procedure is described for correction of this deformity. In principle it consists of: 1. elongating the fissure at the lateral canthus, 2. correcting the fold of skin overhanging the medial canthus, 3. deepening or creating a medial canthal depression, 4. moving the medial canthus, and, if necessary, the lower lacrimal punctum, nasally, and at the same time creating as much of a depression in this area as possible, and 5. at a second stage, elevating and creating a fold in the upper lid. (8 figures) G. S. Tyner.

Isola, W., Borras, A., Nin, C., and Ferrer, J. **Recent advances in the treatment of the lids and lacrimal system.** Arq. brasil. de oftal. **17**:51-88, 1954.

The authors review developments in therapy of the lids and lacrimal system which have appeared within the last four years. Medical treatment of the lids has been little modified with the exception of the use of new antibiotics and hydrocortisone. The difficulties in treating blepharitis are considered, and the possible etiology of allergy and mycotic infection should be considered.

Ptosis surgery has been advanced by Berke's technique which is highly recommended, since it results in few cases of vertical imbalance although the superior rectus muscle is used.

Many procedures have been reported for the treatment of ectropion and entropion—too many to be reviewed.

In diseases of the lacrimal system, most advances have been made in treating chronic dacryocystitis. Those cases which are primarily of endocrine origin respond most satisfactorily to prolonged irrigation with neostigmine. Less satisfactory results are obtained with estrogens. Antibiotics, astringents and vasoconstrictors have their place, but when these are not successful, surgery becomes necessary. Dacryocystorhinostomy is the procedure of choice, and most authors prefer the external approach. A variety of techniques is described. (220 references)

James W. Brennan.

Jones, I. S., and Pfeiffer, R. L. **Lacrimal gland tumors. Roentgenographic diagnosis.** Tr. Am. Acad. Ophth. **58**:841-847, Nov.-Dec., 1954.

The authors present twenty cases of lacrimal gland tumor with 16 photographs of the roentgen films and three tables. They discuss briefly the X-ray features of bony changes from the standpoint of diagnosis, differential value and prognosis. (17 figures, 3 tables, 14 references)

Theodore M. Shapira.

Reeh, M. J., and Hyman. S. **Treatment of lid tumors.** Tr. Am. Acad. Ophth. **59**: 507-521, July-Aug., 1955.

The authors discuss the anatomy of the lid and the various procedures for dealing with lid tumors and their merits and their limitations. (15 figures, 13 references)

Theodore M. Shapira.

Romanes, G. J. **Dacryocystorhinostomy —clinical report of fifty cases.** Brit. J. Ophth. **39**:237-240, April, 1955.

Fifty patients treated for epiphora by means of dacryocystorhinostomy are reviewed. Dacryocystograms were made before and after surgery to visualize the site of blocking as well as confirm the presence of an anatomical opening. In the surgical technique several modifications are worth noting. To obtain a good and imperceptible scar the skin incision is made as far forward as possible on the side of the nose. In order to facilitate the operation as well as to avoid the angular vessels, a rhinostomy lamp is inserted for transillumination before and during the operation. The vessels are apparently easily seen by this method and their outline is then marked on the skin by means of ink. The lamp makes it easier to recognize the trephine site and also to remove all bone from the trephine site before incision of the mucous membrane takes place. With the light inside the nose, bleeding points are easily seen. The author stresses the use of hypotensive anesthesia in order to avoid a bloody field and to reduce the need for suction in the area. A number 3 Jacques catheter is particularly important since it is inserted into the fenestra and kept in place for one week. The results were good; 90 percent of patients were relieved of symptoms. Results in children are unpredictable because of abnormal healing and vigorous growth of scar tissue. (1 figure, 1 table, 4 references)

Lawrence L. Garner.

Wies, Frederick A. **Spastic entropion.** Tr. Am. Acad. Ophth. **59**:503-506, July-Aug., 1955.

The author reviews the common methods of treatment of spastic entropions and then describes his own very simple office method. (1 figure, 10 references)

Theodore M. Shapira.

16
TUMORS

Clarke, Edwin. **Ophthalmological complications of multiple myelomatosis.** Brit. J. Ophth. **39**:233-236, April, 1955.

The ophthalmologic aspects of multiple myelomatosis are reviewed and a single case is described. The ocular complications may be the presenting feature of the disease, but usually evidence of generalized myelomatosis is noted. The ophthalmic manifestations may be: 1. vascular retinal changes (retinal hemorrhages due to thrombotic incidents), 2. papilledema (due to cranial extensions of the tumors) 3. ocular nerve palsies (the commonest sequel of invasion of the base of skull), 4. orbital myelomata and 5. lesions of optic nerve pathways. In the case presented diplopia was the only ocular complaint of a patient who had had medical care for two years. The diagnosis was readily made by means of X-ray examination of the skull and long bones. Biopsy of the bone marrow and Bence-Jones study of the urine are of added value in the diagnosis. Therapy is not effective. (1 table, 32 references)

Lawrence L. Garner.

Deering, Donald W. **Basosquamous-cell carcinoma of the orbit.** A.M.A. Arch. Ophth. **54**:428-431, Sept., 1955.

A basosquamous-cell carcinoma of the orbit resulted from fulguration of skin lesions of the eyebrow and cheek. This case demonstrated the need for biopsy of such lesions. The Krönlein procedure was successfully employed for treatment of the orbital lesion. (7 figures, 3 references)

G. S. Tyner.

Donahue, Hugh C. **An exceptional lesion of the orbit.** A.M.A. Arch. Ophth. **54**:259-261, Aug., 1955.

This report gives evidence of the benignity of glioma of the optic nerve. A massive recurrence developed during the 22 years after enucleation and excision of a portion of the optic nerve. After 22 years there was no intracranial extension or metastasis elsewhere in the body. (1 figure, 5 references) G. S. Tyner.

Espidora-Couso, José. **Kahler's myelomatosis.** Arch. chil. de oftal. **12**:85-88, Jan.-June, 1955.

A patient with Kahler's myelomatosis had bilateral blindness without optic disc changes associated with severe headaches and paralysis of all the cranial nerves except the first and eighth. There was a destruction of the bony foramina. (2 figures, 3 references) Walter Mayer.

Gareis, Richard. **Epithelial tumors of the ciliary body.** Klin. monatsbl. f. Augenh. **127**:344-358, 1955.

The author describes a diktyoma which occurred in the left eye of an 18-months-old child and a malignant medullo-epithelioma in a 45-year-old woman. (5 figures, 37 references) Frederick C. Blodi.

Gärtner, J. **Histogenesis of orbital reticulum cell sarcoma.** Klin. Monatsbl. f. Augenh. **127**:335-344, 1955.

A 63-year-old patient had a severe exophthalmus on the right side for more than a year. The eye became blind and an exenteration was done. X-ray study of the orbit showed no pathologic changes. Two tumor nodules were found consisting of reticulum cells. The tumors were adjacent to extraocular muscules where orbital lymph vessels have recently been found (Hayashi, Nishimura). (3 figures, 26 references) Frederick C. Blodi.

Malone, Roos G. S. **Dictyoma.** Brit. J. Ophth. **39**:429-436, July, 1955.

The clinical and pathologic findings in a case of dictyoma are described and dis-

cussed extensively. The tumor recurred after exenteration of the orbit and invaded skull and brain. (6 figures, 28 references)

Morris Kaplan.

Schrire, Louis. **Ossification in a conjunctival tumour.** Brit. J. Ophth. **39**:443-445, July, 1955.

A three-year-old girl is described in whom a conjunctival tumor, which may have been a fibroma or a dermoid, became ossified. (3 figures, 2 references)

Morris Kaplan.

Whorton, C. M., and Paterson, J. B. **Carcinoma of Moll's glands with extramammary Paget's disease of the eyelid.** Cancer **8**:1009-1015, Sept.-Oct., 1955.

A case of carcinoma of Moll's gland of the right upper lid with intraepithelial extension to the surface skin of the lid margin is described. The lesion was moist, weeping, and superficial, of slow progression and resistant to therapy. Finally an exenteration of the orbit became necessary to effect a cure. (6 figures, 20 references)

Irwin E. Gaynon.

17

INJURIES

Bellavia, M. **The ocular effects of intoxication with illuminating gas.** Arch. di ottal. **59**:237-245, May-June, 1955.

Three cases were observed where the only eye sign after accidental inhalation of gas was a slight blurring of vision. The retinal arterial pressure was slightly elevated for several days. The same findings were encountered in six rabbits after experimental inhalation of illuminating gas. (11 references)

John J. Stern.

Doggart, James Hamilton. **The impact of boxing upon the visual apparatus.** A.M.A. Arch. Ophth. **54**:161-169, Aug., 1955.

In an interesting and amusing way, the author points out the often disastrous effects of boxing upon the visual apparatus. (15 references)

G. S. Tyner.

Landesberg, Jacques. **Chorioretinitis produced by atomic bomb explosion.** A.M.A. Arch. Ophth. **54**:539-540, Oct., 1955.

Retinal burns can result from improper shielding of the eyes from an atomic blast even at distances greater than those resulting in skin burns. The fireball of an atomic blast is 100 times as bright as the sun. A case of retinal damage is reported in a soldier who "sneaked" a look at a tower blast. (2 references)

G. S. Tyner.

Marsico, Vincenzo. **A case of penetration and retention of eyelash in the anterior chamber.** Arch. di ottal **59**:13-21, Jan.-Feb., 1955.

An eyelash was found implanted in the anterior chamber after a perforating injury. After healing of the corneal laceration the eye remained irritated. The eyelash was removed through a corneal incision and uneventful recovery (final vision 20/20) followed. (9 references)

John J. Stern.

Masuda, Y. **Recent status of atomic bomb cataract in Hiroshima.** Acta Soc. Ophth. Japan **59**:899-906, July, 1955.

During the past two years 525 persons injured as a result of the atomic bomb explosion in Hiroshima were examined at random during the past two years, namely, eight to ten years after the explosion. In 138 of the 520 persons or in 27 percent, atomic bomb cataract was demonstrated. The cataract was found more frequently in younger people than in the aged; namely, 36 percent of those 10 to 15 years of age, 28 percent 16 to 25 years of age, 26 percent 26 to 40 years of age, and 16 percent over 41 years of age. Most of

these persons suffered from the exposure within the radius of 2 km. from the explosion center. Severe cataract was observed in persons who had shown other atomic bomb symptoms such as loss of hair, bleeding, diarrhea and fever. About half of these cataract patients had a vision of 20/20. In four of the 138 cases, there was a reduction of vision below 20/200 by cataract. (1 figure, 1 table, 20 references) Yukihiko, Mitsui.

Páez Allende, Francisco. **Fundus changes in a case of chest and pelvis compression (Purtscher's disease).** Arch. oftal. Buenos Aires **30**:140-146, April, 1955.

Immediately after a severe compression injury of the head, chest, and pelvis, which caused a fracture of the latter, the patient, a 41-year-old man, noticed a sudden loss of vision of the left eye, in the fundus of which some hemorrhages would have been detected in the following days. When first examined, a year later, hemorrhages were still present in the left eyeground, one of which could be seen at the macula; in addition, there was marked pallor of the disc. Vision with the affected left eye, first reduced to 4/100, improved subsequently to 20/200. Although the pathogenesis of the ophthalmic changes occurring in such cases is still far from clear, it seems highly probable that they represent a particular form of Trueta's crush syndrome (cf. Weinstein, P.: Am. J. Ophth. 36:660-662, 1953). (1 figure. 21 references) A. Urrets-Zavalia, Jr.

Passow, Arnold. **An early operation for chemical injuries of the eye.** Klin. Monstbl. f. Augenh. **127**:129-142, 1955.

This operation was originally described in 1938 and not to be performed in hospital. It should be done early and in the hands of the author it has brought about healing or prevented the development of corneal opacities and necrosis. The conjunctiva is incised along the limbus and mobilised. The ischemic or chemotic conjunctiva is undermined and only completely necrotic parts are excised. Two triangular pieces are then excised and the conjunctiva is sutured in such a way that it covers the entire corneal periphery. (4 figures, 7 references) Frederick C. Blodi.

Spaeth, E. B., Fralick, F. B., and Hughes, Jr., W. F. **Estimation of loss of visual efficiency.** Arch. Industrial Health **12**:439-449, Oct., 1955.

The committee appointed by the Council on Industrial Health, American Medical Association, submitted its revision of the Appraisal of Loss of Visual Efficiency and it was approved and accepted by the Executive Committee of the Section on Ophthalmology. The report provides a description of the methods of determining the loss of visual efficiency of a person who has suffered visual impairment as a result of disease or injury. (7 figures, 2 tables, 1 reference) F. H. Haessler.

18

SYSTEMIC DISEASE AND PARASITES

Albeaux-Fernet, M., Guiot, J., Braun, S., and Romani, J. D. **Results of surgical hypophysectomy in a case of malignant edematous exophthalmos** J. Clin. Endocr. and Metab. **15**:1239-1256, Oct., 1955.

In a patient in whom malignant exophthalmos was aggravated by treatment of the associated hyperthyroidism, marked amelioration of the local and general disturbances was produced by surgical hypophysectomy. (3 figures, 2 tables, 6 references) Irwin E. Gaynon.

Cascio, G., and Ponte, F. **The ocular complications of brucellosis.** Rassegna ital. d'ottal. **24**:194-201, May-June, 1955.

The authors discuss brucellosis and report a case of brucella uveitis. They ex-

plain the verification of the diagnosis, the complications and the pathogenetic process. With observations drawn from the literature they document the possibility that optic neuritis and the paralysis of the extrinsic muscles are not secondary to a generalized infection but to one of the central nervous system, that is, a neurobrucellosis. (48 references)

Eugene M. Blake.

Contardo, René. **Diseases of collagenous tissue.** Arq. brasil. de oftal. **17**:114-126, 1954.

The concept of disease involving connective tissue is ancient, dating to the time of Morgagni, although attention was called to callagenous diseases as such in 1942. Since connective tissue is disseminated throughout the body, the common denominator is a protein derived from mesenchyme collagen. Because of the abundant connective tissue in the eye and adnexa, it is necessary to understand the various diseases of collagenous tissue which may have ocular manifestations. Some of the diseases are serum sickness, disseminated lupus erythematosus, periarteritis nodosa, rheumatoid arthritis, scleroderma, dermatomyositis and temporal arteritis. A vascular disturbance is fundamental in these diseases, most evident in generalized lupus erythematosus and periarteritis nodosa. Observation of ocular lesions assist one in evaluating the status of the disease in other organs and the eye may become involved earlier in the course of the disease.

The author reports his observations in disseminated lupus erythematosus, scleroderma, temporal arteritis and rheumatoid arthritis. Uveitis was most commonly present in the patients with arthritis. Keratitis, scleritis, and retinopathy were also noted. A tendency toward recurrence and resistance to treatment characterized the ocular lesions. (2 figures, 1 table, 79 references) James W. Brennan.

Dernoncourt, Y. **Behçet's syndrome.** Ann. d'ocul. **188**:556-571, June, 1955.

Two cases of Behçet's syndrome are reported. In one, a 25-year-old girl, the ocular, buccal, and genital lesions recurred with each menstruation. In the other, a 23-year-old boy, there was an associated transient hydrarthrosis of the wrist joints. (57 references) John C. Locke.

Gemolotto, Gugielmo. **Contribution to the surgical treatment of intraocular cysticercus.** Arch. di ottal. **59**:365-375, Sept.-Oct., 1955.

An intraocular cysticercus was successfully removed through a scleral incision in an area prepared by diathermy coagulation after careful localization of the parasite and under ophthalmoscopic control. (61 references) John J. Stern.

Guillaumat, L. **Ophthalmoscopy and puerperal eclampsia.** Union Med. Canada **84**:1143-1149, Oct., 1955.

In the toxemic retinopathy of pregnancy the retinal changes run parallel with the severity of hypertension. The first visible ocular sign is an attenuation of the retinal arterioles and a rise in the diastolic pressure of the central retinal artery as shown by the Bailliart dynamometer. The ratio of this diastolic pressure to that of the brachial artery, normally slightly less than 50 percent, is much greater in the toxemia of pregnancy. This difference is constant, early and fundamental. The article contains an excellent digest of the literature, but unfortunately lacks a bibliography.

James E. Lebensohn.

Kruemmel, H., and Rausch, L. **Pseudoglioma in a case of pigment dermatosis of**

Siemens-Bloch. Ophthalmologica **130**:31-53, July, 1955.

The pigment dermatosis of Siemens and Bloch, when first described in 1925, was thought to be a rare congenital anomaly characterized by inability of the melanoblasts of the basal cells in the epidermis to retain their pigment ("incontinentia pigmenti"). More recent studies showed this incontinence to occur in other dermatoses. The congenital form of the disease is rare, but has been consistently associated with ocular anomalies. In the case under review the ocular anomaly consisted of a unilateral developmental anomaly characterized by persistence of the hyaloid artery and of the primary vitreous, producing the clinical picture of pseudoglioma. The authors suggest more than just a coincidental relationship between the dermatosis and ocular anomaly. (17 figures, 1 table, 29 references)

Peter C. Kronfeld.

Lee, R. E. **Hemodynamic changes in the bulbar conjunctival capillary bed of subjects with hypertension associated with "Cushing's Syndrome" or pheochromocytoma.** Am. J. Med. **19**:203-208, Aug., 1955.

Five patients with Cushing's disease, and three with pheochromocytoma had biomicroscopic examination of the bulbar conjunctival vessels. The author had previously described criteria in four aspects in normal subjects, those with essential hypertension, and those with normotensive disease. The four criteria concern vasoconstriction of the minute terminal arterioles and metarterioles; increased reactivity to epinephrine; reduction in velocity of peripheral blood flow; and changes in vascular topography, such as elongation, coiling, and tortuosity. It was found that the observations in cases of Cushing's disease were similar to those in essential hypertension; but those in pheochromocytoma were similar to normotensive cases, except in hypertensive crisis, when the findings exceeded those of essential hypertension. (3 tables, 4 figures, 3 references) Harry Horwich.

Leo, Margot. **Ocular changes in pulseless disease.** Klin. Monatsbl. f. Augenh. **127**:284-294, 1955.

The author describes two cases. The first patient was a 42-year-old woman. The retinal veins were dilated and sausage-shaped. There were arteriovenous shunts on both discs and granular circulation in the veins. In the fundi were numerous small hemorrhages and the pressure in the retinal arteries was remarkably low.

The second patient was a 41-year-old woman with numerous neurologic signs. Both fundi showed, after the cataracts were extracted, narrow retinal vessels which did not reach the periphery but returned as arteriovenous anastomoses. There was also an unusual vessel in the chamber angle. (4 figures, 18 references)

Frederick C. Blodi.

Maeder, G., and Forni, S. **Ocular manifestations of pheochromocytoma.** Ophthalmologica **129**:330-334, April-May, 1955.

A typical case of pheochromocytoma in a six-year-old boy showed, ophthalmoscopically, a type four neuroretinopathy of Keith and Wagener. The retinal as well as all other tangible manifestations of the disease disappeared almost completely after surgical removal of two pheochromocytomas located at the hilus of the left kidney. (13 references)

Peter C. Kronfeld.

Rosselet, Ed. **Arterial occlusion syndromes.** Ophthalmologica **129**:321-325, April-May, 1955.

Four cases of obliterating arterial disease with prominent eye findings are reported. Two of the cases were of the

temporal arteritis type. In one case unilateral sudden occlusion of the central artery was part of a thrombosis of the common carotid secondary to a luetic aneurysm of the aorta. In the fourth case, a man, 54 years of age, first developed circulatory difficulties in his lower extremities which were partly relieved by sympathectomy. Several years later he was readmitted with an acute surgical abdomen which proved to be due to thrombosis of one renal artery. Ophthalmologically he showed marked sclerosis of the retinal arterioles and marked discrepancy between the general (200/95) and the retinal blood pressure. This discrepancy, that is a state of retinal hypotension, is an important symptom of vascular diseases with specific localization in the carotid and ophthalmic arteries.

Peter C. Kronfeld.

Rougier, J., and Garin, J. P. **Ocular toxoplasmosis.** Ann. d'ocul. **188**:493-534, June, 1955.

The authors report twelve cases, which they believe to be examples of acquired ocular toxoplasmosis. Three patients had juxtapapillary choroiditis, three disseminated choroiditis, three uveitis, and three iridocyclitis. A study of the literature of the past five years makes it clear that acquired ocular toxoplasmosis does exist. Diagnosis is established by the absence of other positive etiologic findings, and by an evaluation of the changes in antibody titer disclosed by the complement-fixation test. In acquired toxoplasmosis, the antibody titer is at first rising and later falling. (4 figures, 76 references)

John C. Locke.

Sinskey, R. M., and Anderson, W. B. **Miliary blastomycosis with metastatic spread to posterior uvea of both eyes.** A.M.A. Arch. Ophth. **54**:602-604, Oct., 1955.

Choroidal granulomas are reported as a finding at autopsy in a 39-year-old negro with this disease. (3 figures, 1 reference)

G. S. Tyner.

19

CONGENITAL DEFORMITIES, HEREDITY

Alper, M. G., and Dessoff, J. **Porencephaly.** A.M.A. Arch. Ophth. **54**:541-547, Oct., 1955.

Porencephaly is a relatively rare condition characterized by a defect or cavity in the brain which often communicates with the ventricular system and subarachnoid space and is filled with cerebrospinal fluid. The ocular manifestations in the reported case are a left homonymous hemianopsia with macular sparing, from which the patient recovered spontaneously, and visual hallucinations. Operative interference in this condition is contraindicated if communication exists with the ventricular system and subarachnoid space. (7 figures, 12 references)

G. S. Tyner.

Forsythe, W. I., **Congenital hereditary vertical nystagmus.** J. Neurol., Neurosurg. & Psychiat. **18**:196-198, Aug., 1955.

A three-generation pedigree of twelve members of an Italian family is presented. Five out of the twelve had congenital hereditary vertical nystagmus. Apparently this is the first report of this condition. The pedigree with linkage data shows no consistent relationship in other factors, such as sex, color of eyes, blood group, taste, and labyrinthine reaction. (1 chart, 13 references) Harry Horwich.

Piffaretti, A. **Correlations between ocular malformations.** Ophthalmologica **129**: 342-346, April-May, 1955.

The cases of ocular malformations seen at the Eye Clinic of the University of Lausanne were studied statistically in

order to determine the relationship between two or more coexisting malformations. As far as can be judged from the short communication, no definite conclusions were reached. (4 tables)

Peter C. Kronfeld.

Salorio, Manuel Sanchez. **Syndrome of a disturbance in the development of the anterior segment of the ectoblast. (Retinitis pigmentosa, cataract, anisocoria, palpebral hypotricosis associated with oligophrenia and somatic hypoevolution.)** Arch. Soc. oftal. hispano-am. **15**:496-508, May, 1955.

This is a report of a case of a 16-year-old boy, whose parents were cousins, with an intelligence quotient of 60, who had an atrophy of the skin of the lids, loss of eyelashes, anisocoria, immature cataracts, and retinitis pigmentosa. The author points out that this is a clinical syndrome indicative of a hereditary lesion, recessive in character, caused by a disturbance in development at the time of differentiation of the anterior segment of the ectoblast. Such a lesion explains the involvement of the cerebral cortex, the retina, the skin, and the lens. (2 figures, 4 references)

Ray K. Daily.

Sbordone, G. **Keratoconus and retinitis pigmentosa.** Arch. di ottal. **59**:211-219, May-June, 1955.

Four brothers, the offspring of a marriage between niece and uncle, had keratoconus, retinitis pigmentosa and oligophrenia. No precedents could be found in the family. Two of the living children have normal eyes, and three deceased children were stated to have had severe eye trouble. Various genetic hypotheses are discussed. (10 references)

John J. Stern.

Scott, J. G., Friedmann, A. I., Chitters, M., and Pepler, W. J. **Ocular changes in the Block-Sulzberger syndrome (incontinentia pigmenti).** Brit. J. Ophth. **39**: 276-282, May, 1955.

The Block-Sulzberger syndrome is a rare, probably familial, disease characterized by abnormality of development of tissues arising from ectoderm. The defects are found in skin, nails, hair, teeth, central nervous system and eyes, and chiefly in girls soon after birth. In the skin a characteristic inflammatory eruption in a definite linear pattern occurs. Only 91 cases have been reported and in 23 of them there were ocular defects. Another case is added; a newborn infant girl in Johannesburg presented the typical skin lesions and both eyes were defective. She seemed to have fair vision at first, but one eye rather quickly developed phthisis bulbi and the other became inflamed. This eye was subsequently enucleated at the age of two years. The eyeball showed little that was pathologic except a total funnel-shaped detachment of the retina. In several other reported cases this mass in the fundus was mistaken for metastatic endophthalmitis, pseudo-glioma, and retrolental fibroplasia. (6 figures, 50 references)

Morris Kaplan.

Sorsby, A., and Davey, J. B. **Dominant macular dystrophy.** Brit. J. Ophth. **39**: 385-397, July, 1955.

Dominant macular dystrophy is much less easily recognized than the recessive macular dystrophy of the Stargardt type since the signs and symptoms can remain silent for years. It is, however, a definite clinical entity and the authors described its occurrence in three families. There were seven cases in three generations in one branch of one family and two cases in two generations in the other. The members of this family show considerable variation in loss of visual acuity, but all showed about the same loss of color vision. The second family had three af-

fected members in two generations; two sisters presented the same picture while the daughter of one had much more loss of vision. The third family had four affected members in three generations and again all showed about the same picture. The ophthalmoscopic picture in these cases closely resembles that found in the recessive type of the disease but the difference lies in its very slow onset, probably years before it can be seen clinically, while in the Stargardt type the onset is quite sudden with rapid progression. Serious loss of red-green vision is common to both and these conditions must be differentiated from congenital total color blindness. (11 figures, 3 charts, 18 references)

Morris Kaplan.

Sorsby, A., and Davey, J. B. **Generalized choroidal sclerosis.** Brit. J. Ophth. **39**: 257-275, May, 1955.

Two families in which generalized choroidal sclerosis occurred are described with some detail; one family presented had four affected members in two generations, the other ten in two generations. In the first family, the disease began late in life, remained very mild and affected all about equally; in the second family some were affected in their twenties and some had very serious loss of vision. The disease begins with edema and hemorrhage of the choroid which brings about mottling of the whole area. The central choroidal vessels become exposed rather rapidly. They become sclerosed and are then seen as bright white streaks radiating out from the disc and they are crossed by normal retinal vessels. The whole central portion of the choroid then atrophies and then the sclera is clearly visible. Bone corpuscle pigment occurs, but this is central rather than peripheral. The absence of night blindness distinguishes this anomaly from retinitis pigmentosa. This study suggests that generalized choroidal sclerosis is a dominant hereditary characteristic whereas retinitis pigmentosa is recessive. (13 figures, 5 references)

Morris Kaplan.

Tower, Paul. **The fundus oculi in monozygotic twins.** A.M.A. Arch. Ophth. **54**: 225-239, Aug., 1955.

Studies of the fundus oculi of 6 pairs of identical twins disclosed a close similarity between all structures except the distribution of retinal vessels. (6 figures, 42 references)

G. S. Tyner.

Victoria, V. **Leber's disease. Report of an affected family.** Arch. oftal. Buenos Aires **30**:249-256, July, 1955.

In a family of 15 persons in four generations, hereditary optic atrophy occurred in three male and three female subjects. While in the former the condition became manifest at the ages of 12, 13 and 20 years, in the latter it was acquired in early childhood. In opposition to the classical, recessive, sex-linked form where it appears largely in men and is carried only by women, according to Lossen's law, in the present pedigree the disease was seen to follow an apparently dominant, autosomal pattern.

As to the perennial question of whether, in the presence of such a mode of inheritance, the diagnosis of Leber's disease would be justified, it may be well to remember that in Lundsgaard's opinion the condition is always transmitted autosomally, as a dominant trait, its penetrance, however, being much more marked in men than in women (Lundsgaard, R.: Leber's disease. Acta Ophthalmologica Suppl. 21, 1944). On the other hand, Sorsby's judicious words on the subject deserve special mention: "It would appear that excessive attention has been paid to the Leber type of optic atrophy and its difficult mode of inheritance as to the one and only possible

variety of abiotrophic optic atrophy . . . it is not unlikely that several modes of inheritance, and several clinical types of hereditary optic atrophy exist. . . ." (Sorsby, A.: Genetics in Ophthalmology. London, Butterworth, 1951). (1 chart, 6 figures) A. Urrets-Zavalia, Jr.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

v. Herrenschwand, F. **The influence of light stimuli on the vegetative control of the metabolism.** Klin. Monatsbl. f. Augenh. **127**:219-220, 1955.

The author remembers a nine-year-old boy who had bilateral cataract and was operated on 44 years ago. After the successful operation the child improved mentally and physically. This is thought to be a proof of Hollwich's hypothesis that visual stimuli influence the metabolism. (9 references) Frederick C. Blodi.

Leggo, Christopher. **A second look at one-eyed applicants and employees.** Indust. Med. **24**:473-476, Nov., 1955.

Although one cannot say that all one-eyed applicants will make good employees it is equally fallacious to say that they will not. To deny employment to the many members of the large group of the one-eyed is an unsound and unjust policy. There is no longer a reasonable risk under compensation laws for a second injury and the policy may be modified to benefit not only the one-eyed but also their potential employers and the community in which they live. F. H. Haessler.

Nataf, Roger. **In regard to the circulation of the lymphatics of the conjunctiva.** Arch. d'opht. **15**:402-403, 1955.

The author refers to his article with Delon (Bull. Soc. Fr. d'opht., 1953, p. 171) in which original observations were made on the circulation in the lymphatics of the bulbar conjunctiva by means of staining with Pyrotrope blue. He takes issue with Busacca with respect to his claim of priority in making these observations, and calls attention to the absence in Busacca's publications of any mention of the recurrent nature of the circulation. Phillips Thygeson.

Stephenson, R. W. **The assessment of eye injuries in industry.** Tr. Ophth. Soc. U. Kingdom **74**:515-518, 1954.

The author is helping to form a guide for assessing compensation in eye injuries. He found that injuries to the eye causing more than three days' loss of work form about five percent of all industrial injuries. Central acuity, field of vision and muscle function are taken into account in assessing loss of visual efficiency. Beulah Cushman.

Taieb, Albert. **The memoirs of Casanova and the operation of Ridley.** Arch. d'opht. **15**:501-503, 1955.

The author notes that Jacques Casanova, in addition to conducting his amorous adventures, was a traveller and historian who never neglected an opportunity to consort with the great minds of his time. Medicine interested him especially, and one passage in his memoirs recounts his experience with ophthalmologists and ophthalmic charlatans, particularly in the treatment of cataract. He describes a charlatan who claimed to replace the crystalline lens with a highly polished crystal. This claim was lampooned in a comic article written by a professor in the Faculty of Medicine at Warsaw who likened the replacement of the lens to the replacement of a tooth by a false one. The charlatan, one Tadini by name, who received no support in his own day, may actually have been in advance of his time. P. Thygeson.

NEWS ITEMS

Edited by Donald J. Lyle, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 12th of the month. For adequate publicity, notices of post-graduate courses and meetings should be received three months in advance.

Deaths

Dr. Herman Porter Davidson, Chicago, Illinois, died October 9, 1955, aged 64 years.

Dr. James Lawrence Flynn, Natick, Massachusetts, died September 26, 1955, aged 44 years.

Dr. John Nelson Osburn, Balboa, California, died October 11, 1955, aged 69 years.

Dr. Edna Marguerite Reynolds, Denver, Colorado, died September 25, 1955, aged 65 years.

Dr. James Washington Smith, New York, died October 24, 1955, aged 62 years.

Dr. Will Walter, Charlottesville, Virginia, died in October, 1955, aged 88 years.

Harold Pace Gibb

Mr. H. P. Gibb, surgeon to the Central London Ophthalmic Hospital, who died at Gerrard's Cross on November 21st at the age of 77 years, received his medical training at the University of Cambridge and St. Bartholomew's Hospital. After qualifying in 1904 he went on to take the F.R.C.S. in 1906 and the M.B. Camb. in 1907. J. H. P. writes: "He was a man of so retiring a disposition that his outstanding ability was known to only a few. He was my clinical assistant at Moorfields for several years, coming to me with an established reputation as a neurologist, won as a house-physician at Queens Square. He soon gained an equally high reputation as an ophthalmologist. He took little interest in private practice, but devoted himself wholeheartedly to hospital practice, winning the warm affection of his patients. During the whole of the late war he did most of the work at the Central London Ophthalmic Hospital, which was depleted of the younger members of its staff. He was a brilliant clinician, and it was a matter of regret to me that he did not write a book on the neurologic aspects of ophthalmology. No one was better qualified to do so. He had many interests outside medicine—art, sport, and literature. He was a friend whom I valued very highly; and it has always been a regret to me that he did not attain that wider recognition to which his intellect and character entitled him." (From *The Lancet*, December 3, 1955.)

American Orthoptic Council

Dr. Edmond L. Cooper, 414 David Whitney Building, Detroit 26, Michigan, has succeeded to the secretary-treasurership of the American Orthoptic Council, and all inquiries and correspondence relevant to American Orthoptic Council activities should be addressed to him with the following exceptions:

1. Correspondence relevant to the examinations offered by the American Orthoptic Council, and applications for such examination should be addressed to the chairman on examinations, Dr. Frank D. Costenbader, 1605 22nd Street, N.W., Washington 8, D.C.

2. Correspondence related to the training of orthoptic technicians, and applications for such instruction should be addressed to the chairman of instruction, Dr. Hermann M. Burian, Department of Ophthalmology, Iowa University Hospitals, Iowa City, Iowa.

New York Conference

The New York Society for Clinical Ophthalmology announces a Participating Conference to be held at the Waldorf-Astoria Hotel, New York, Friday and Saturday, April 13 and 14, 1956. Registrants will be able to participate in seminars on various subjects, including glaucoma, biomicroscopy, therapeutics, muscles, and neuro-ophthalmology. The discussions will be led by prominent members of the society. Registration will be limited to 100 and the fee of $35.00 will include luncheons. For further information write to:

Miss Gloria Benabo
737 Park Avenue
New York 21, New York

International Correspondence Society

The International Correspondence Society of Ophthalmologists and Otolaryngologists was launched with the first mailing on January 1, 1956. This correspondence society is an organization whose sole purpose is to publish the loose-leaf collected letters of its membership every two weeks. The letters of the two specialties will be published separately as the *Collected Ophthalmology Letters* and the *Collected Otolaryngology Letters*. The editor will be Dr. Jack R. Anderson, New Orleans, and on the ophthalmology editorial advisory board are: Dr. James H. Allen, New Orleans; Dr. Michael J. Hogan, San Francisco; Dr. P. Robb McDonald and Dr. Harold G. Scheie, Philadelphia; and Dr. Hugh L. Ormsby, Toronto. For further information write to:

International Correspondence Society of
Ophthalmologists and Otolaryngologists
705 Baronne Building
New Orleans 12, Louisiana

Residency available

A three-year approved residency in ophthalmology with affiliated children's training is available at the

Veterans Administration Center, Wood, Wisconsin. The stipend: $2,840, $3,195, and $3,550. For further information write:

R. H. Lehman, M.D., Chief,
Eye, Ear, Nose, and Throat Section,
Veterans Administration Center,
Wood, Wisconsin

NSPB GRANTS-IN-AID

The National Society for the Prevention of Blindness announces the availability of grants-in-aid for basic laboratory or clinical research projects that may add to an understanding of the etiology of blinding eye diseases or lead to improved methods of diagnosis, treatment, or prevention.

To be considered, applications should be received before April 1, 1956. Forms may be obtained from the Research Committee, National Society for the Prevention of Blindness, 1790 Broadway, New York 19, New York.

In December, the National Society reported new research grants amounting to $12,200. These were allotted to:

Dr. Gordon E. Gibbs, University of Nebraska Medical School, Omaha, $4,000, for studies of the adrenocorticoid function in relation to diabetic retinitis.

Dr. Jerry H. Jacobson, New York Eye and Ear Infirmary, $1,500, electrophysiology of the eye.

Dr. Irving H. Leopold, Wills Eye Hospital, Philadalphia, $2,500, choroidal circulation.

Dr. A. Gerard DeVoe and Dr. Robert S. Coles, New York University Post-Graduate Medical School, $1,900, use of pancreatic dornase in inflammatory ocular infections.

Dr. Daniel G. Vaughan, Jr., University of California School of Medicine, San Francisco, $300, evaluation of antibacterial agents against pseudomonas aeruginosa.

Dr. Peter C. Kronfeld, Illinois Eye and Ear Infirmary, Chicago, $2,000, cardiovascular effects upon the human tonogram.

NATIONAL COUNCIL GRANTS-IN-AID

The National Council to Combat Blindness, Inc., 30 Central Park, South, New York 19, New York, in accordance with its program of financing research in ophthalmology and its related sciences, announces its 1955-56 awards for grants-in-aid and fellowships, as approved by its Scientific Advisory Committee.

Grant-in-aid. Retina Foundation, Boston, Endre A. Balazs, M.D. (continuation) ($3,800); Synthesis of hyaluronic acid in the vitreous body of embryos and young animals.

New York University-Bellevue Medical Center, New York; Goodwin M. Breinin, M.D. ($2,300); Electromyography of the extraocular muscles including stimulation studies and evaluation of drug effects on the action currents.

State University of Iowa, Iowa City, Iowa, Hermann M. Burian, M.D. (continuation) ($500); Studies of the human electroretinogram.

New York Medical College, Flower and Fifth Avenue Hospitals, New York; Benjamin Friedman, M.D. ($1,250); The identification of lens-dissolving enzymes in aqueous humor and lens.

New York Hospital-Cornell Medical Center, Dan M. Gordon, M.D. ($4,000); Application of visual aids in patients with subnormal vision.

University of California at Los Angeles; S. Rodman Irvine, M. D., Robert Brunish, M.D. ($4,550); Hyaluronic acid and hyaluronidase in ocular tissue.

New York Eye and Ear Infirmary, New York; Jerry Hart Jacobson, M.D. (continuation) ($2,100); Electroretinography in retinal diseases.

University of California, Francis I. Proctor Foundation, San Francisco, Samuel J. Kimura, M.D. (continuation) ($2,000); Study of keratitis sicca and Sjøgren's syndrome.

Wills Eye Hospital, Philadelphia; Harry Green, Ph.D., Irving H. Leopold, M.D. ($4,500); Intraocular pressure and bicarbonate concentration in animal eyes.

State University of Iowa, Iowa City, Iowa; P. J. Leinfelder, M.D. (continuation) ($1,971); The effect of change in pH in the aqueous on metabolism of the lens.

Hadassah University Hospital, Jerusalem, Israel, I. C. Michaelson, M.D. (continuation) ($2,470); (1) Factors affecting new vessel growth in the cornea. (2) Changes in hyaluronic-acid content of vitreous in physiologic and pathologic conditions.

Kumamoto University Medical School, Japan; Yukihiko Mitsui, M.D., (continuation) ($600); Cultivation of trachoma virus in cultivated human cells.

University of Oxford, Nuffield Laboratory of Ophthalmology, England; Antoinette Pirie, Ph.D. ($2,000); The study of changes in the constituents and metabolism of the lens during development of cataract.

Tulane University, Louisiana School of Medicine, New Orleans; J. William Rosenthal, M.D. ($600); A genetic and ophthalmic study of a family showing acromegaly, unusual abnormalities of the skin, and corneal dystrophy.

College of Physicians and Surgeons, Columbia University, New York; George K. Smelser, Ph.D. (continuation) ($1,500); Investigation of experimentally produced exophthalmos.

Manhattan Eye, Ear and Throat Hospital, New York; C. C. Teng, M.D. (continuation) ($1,000): Optic-nerve study.

Okayama University Medical School, Japan, Jun Tsutsui, M.D. ($1,000); Clinical and virologic studies on trachoma especially immunity, spontaneous cure and tumorlike nature of the infected tissue.

Biophysics Research Laboratories, University of Pittsburgh; Jerome J. Wolken, Ph.D. (continuation) ($2,600); Photoreceptor structures.

Institute of Ophthalmology, University of London, England; Alan Woodin, Ph.D. ($1,800); Clarification of the cause of resistance to outflow of the aqueous humor with particular reference to simple glaucoma.

Full-time research fellowships. Edgar Auerbach, M.D., Harvard University, the Biological Laboratories. George Wald, Ph.D., Director, Direct Supervisor ($4,800). Color vision, dark, and light adaptors.

Norman I. Krinsky, Ph.D., Harvard University, the Biological Laboratories, George Wald, Ph.D., Director, Direct Supervisor ($3,600). Esterification of vitamin A by tissue of the eye.

Ralph Levine, M.D., New York University-Bellevue Medical Center. Goodwin M. Breinin, M.D., Director of Research and A. Gerard DeVoe, M.D., Chairman, Department of Ophthalmology, Direct Supervisors ($3,600). The use of radioactive isotopes in ophthalmology.

Part-time research fellowship. Albert B. Chatzinoff, M.D., Mount Sinai Hospital, New York. Frederick H. Theodore, M.D., Acting Chief, Department of Ophthalmology, Direct Supervisor ($1,500). Chronic deficiency of 1-cis vitamin A as the possible etiology of retinitis pigmentosa.

Summer fellowships. Donald Able Berman, Tulane University School of Medicine, New Orleans, James H. Allen, M.D., Chief Department of Ophthalmology, and Marion A. Guidry, M.D., Direct Supervisors ($600). Studies on the electrophoretic properties of soluble corneal proteins.

Arthur Berken, Washington University School of Medicine, Saint Louis, Bernard Becker, M.D., Chief, Department of Ophthalmology, Direct Supervisor ($400). The effect of cortisone administration on the cells of the islets of Langerhans and the capillaries of the pancreas.

George Contis, Biophysics Research Laboratory, University of Pittsburgh, Jerome J. Wolken, Ph.D., Director, Direct Supervisor ($400). Studies: Drosophila melancaster.

Carl Gates Freese, Jr., M.D., Massachusetts Eye and Ear Infirmary, Boston, David G. Cogan, M.D., Direct Supervisor ($400). Evaluation of the syndrome known as "Spasm of the near reflex."

Barbara B. Green, University of California, Phillips Thygeson, M.D., Direct Supervisor ($600). Cytology of the aqueous in experimental uveitis.

Arthur Robert Kallmann, University of California, Michael J. Hogan, M.D., Direct Supervisor. ($600). Histologic studies of aqueous drainage into the canal of Schlemm.

Charles A. Perera, M.D., is the newly elected chairman of the Scientific Advisory Committee and will serve in this capacity for a two-year term.

Irving H. Leopold, M.D., Director of Research and attending surgeon at Wills Eye Hospital, Philadelphia, and Chairman of the Department of Ophthalmology of Graduate School of Medicine, University of Pennsylvania; and Bernard Becker, M.D., Professor and Head, Department of Ophthalmology, Washington University, School of Medicine, Saint Louis, have been added to the Scientific Advisory Committee of the organization. Other members are:

James H. Allen, M.D., Chief, Department of Ophthalmology, Tulane University School of Medicine, New Orleans; Alson E. Braley, M.D., Director, Department of Ophthalmology, State University of Iowa, Iowa City, Iowa; Arthur G. DeVoe, M.D., Chief, Department of Ophthalmology, New York University-Bellevue Medical Center, New York; Louis B. Flexner, M.D., Professor, Department of Anatomy, University of Pennsylvania School of Medicine, Philadelphia; Dan M. Gordon, M.D., Assistant Professor of Clinical Surgery, Department of Ophthalmology, New York Hospital-Cornell Medical Center, New York; Charles Haig, Ph.D., Professor, Department of Physiology, New York Medical College, Flower & Fifth Avenue Hospitals, New York; Michael J. Hogan, M.D., Director, Francis I. Proctor Foundation, University of California Medical School, San Francisco.

Peter C. Kronfeld, M.D., Professor, Department of Ophthalmology, University of Illinois Medical School, Chicago; A. E. Maumenee, M.D., Director, Department of Ophthalmology, Johns Hopkins Hospital and University, Baltimore; Karl Meyer, M.D., Associate Professor, Department of Biochemistry, Columbia University College of Physicians and Surgeons, New York; Stuart Mudd, M.D., Professor, Department of Microbiology, University of Pennsylvania School of Medicine, Philadelphia; Lorrin A. Riggs, Ph.D., Professor, Department of Psychology, Brown University, Providence, Rhode Island; Samuel L. Saltzman, M.D., Associate Professor, Department of Ophthalmology, New York Medical College, Flower and Fifth Avenue Hospitals, New York; Kenneth C. Swan, M.D., Director, Department of Ophthalmology, University of Oregon Medical School, Portland; Phillips Thygeson, M.D., Professor, Department of Ophthalmology, Francis I. Proctor Foundation and University of California Medical Center, San Francisco.

Applications for 1956-57 grants-in-aid and fellowship awards (full-time) will be considered at the seventh annual meeting of the Scientific Advisory Committee, to be held in June, 1956. Closing date for receipt of completed applications for these awards is April 15, 1956.

Applications for summer student fellowships will be reviewed in advance of the meeting and such applications should be filed with the office of the organization no later than April 1, 1956.

All applicants for fellowships, full-time or summer, are required to make their own arrangements for suitable research facilities with accredited institutions.

Appropriate forms may be obtained by addressing:

Secretary
National Council to Combat Blindness, Inc.
30 Central Park, South
New York 19, New York.

Ohio State course

A course designed for physicians devoting full or part time to the practice of ophthalmology will be held on March 5th and 6th at the Ohio State

University Union Building, 13th and High Streets, Columbus. The registration fee will be $30.00. The program will include:

Monday, March 5th. Registration at 8:00 A.M. Classes starting at 9:00 A.M. "Evaluation of ophthalmoplegia," Dr. Sayers; "Subnormal vision and its management," Dr. Sloane; "Clinical use of Diamox," Dr. Thorpe. Afternoon classes beginning at 1:00 P.M. "Common problems in refraction," Dr. Sloane; "Application of gonioscopy," Dr. Thorpe; "The refraction of the aphakic and highly ammetropic patient," Dr. Sloane. A dinner meeting will be held with the Columbus Eye Ear, Nose, and Throat Academy at 6:30 P.M., followed by a discussion of "The management of intraocular foreign bodies," Dr. Thorpe.

On Tuesday, March 6th, classes will convene at 8:00 A.M. "Clinical aspects of ocular tumors and simulating diseases," Dr. Havner; "Antimicrobial drugs in tuberculosis therapy," Dr. Browning; "Degenerative macular lesions," Dr. Falls; "Ocular manifestations of sinus and nasopharyngeal lesions," Dr. Saunders. The afternoon program starts at one o'clock with "Differential diagnosis of chorioretinal lesions," Dr. Falls; "Fundamental aspects of retinal vasomotor activity," Dr. Brecher; "Practical applications of embryology in ophthalmology," Dr. Falls.

Societies

Irish meeting

The Irish Ophthalmological Society will hold its annual meeting in Dublin on May 10th, 11th, and 12th. The Montgomery Lecture will be delivered by Dr. M. E. Alvaro of Brazil at five o'clock on Thursday, May 10th, in Trinity College, Dublin.

Omaha all-day session

On Thursday, March 15th, the Omaha Eye, Ear, Nose, and Throat Society and the Nebraska Academy of Ophthalmology and Otolaryngology are holding a joint all-day session. The principal speaker for ophthalmology will be Dr. Harold G. Scheie, Philadelphia, and the principal speaker for otolaryngology will be Dr. George Shambaugh, Jr., Chicago. Members of the neighboring eye, ear, nose, and throat societies have been invited and the Kansas City Eye, Ear, Nose, and Throat Society is attending as a group.

Madrid officers

Newly elected officers of the Madrid Ophthalmological Society are: President, Dr. Manuel Marin Amat; vice-president, Dr. Juan Arjona Trapote; secretary, Dr. Jose Luis del Rio Cabanas; treasurer, Dr. Pedro Tena Ibarra; director, Dr. Gustavo Leoz de la Fuente.

American College of Surgeons

Dr. Harold G. Scheie, Philadelphia, presided over the opening session of the ophthalmology program at the recent Philadelphia sectional meeting of the American College of Surgeons. Presented at this session were: Symposium on recent advances in ophthalmic surgery: "Cataract surgery," John M. McLean, New York; "Glaucoma surgery," Paul A. Chandler, Boston; "Reconstruction of the lids," Wendell L. Hughes, Hempstead, New York. Dr. Charles L. Schepens, Boston, spoke on "Present status of surgery of retinal detachment," and Dr. R. Townley Paton, New York on "Lamellar transplantation."

At the second session, Dr. Albert D. Ruedemann, Detroit, presided over the symposium on the management of exophthalmos: "Exophthalmos due to pathology in the orbit or adjacent structures," Dr. Wilfred E. Fry, Philadelphia; "Endocrine aspects of exophthalmos," Dr. Edward Rose, Philadelphia; "Treatment of exophthalmos from the viewpoint of the internist," Dr. William Jefferies, Cleveland; "Treatment of exophthalmos from the viewpoint of an ophthalmologist," Dr. John W. Henderson, Rochester, Minnesota.

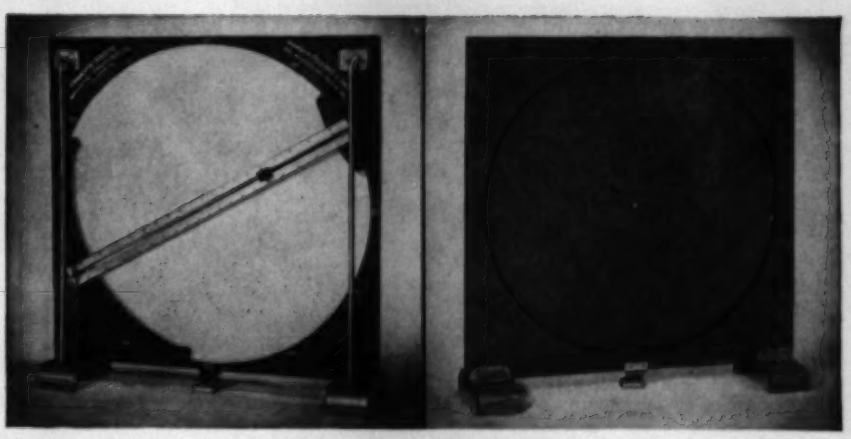
Personals

Dr. Edwin B. Dunphy, Boston, will deliver the annual Charles H. May Memorial Lecture sponsored by the Eye Section of the New York Academy of Medicine, at the Academy on March 19th. The subject of Dr. Dunphy's address will be "Radioactive isotopes in ophthalmology."

Dr. Trygve Gundersen, Boston, and Dr. Jack S. Guyton, Detroit, will be honor guests at the Spring Clinical Conference of the Dallas Southern Clinical Society, March 12th, 13th, and 14th.

Dr. Alson E. Braley, head of the Department of Ophthalmology, Iowa State University, has been appointed chairman of the Ophthalmology Training Grant Committee, National Institute of Neurological Diseases and Blindness of the National Institutes of Health. A distinguished teacher, Dr. Braley will direct and develop a training program in ophthalmology, to reduce the shortage of clinical teachers and investigators through grants made to teaching institutions. The current supply of teachers, research workers, and clinicians is seriously inadequate to meet the required needs for training in this field.

The appointment of Dr. Derrick Vail, Editor-in-Chief and Managing Director of The American Journal of Ophthalmology, to the National Advisory Neurological Diseases and Blindness Council, for a term of one year, has been announced by Surgeon General Leonard A. Scheele of the Public Health Service, U. S. Department of Health, Education, and Welfare. He will serve the unexpired term of the late Dr. Jonas S. Friedenwald of Baltimore, Maryland.



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The Conover-Spaeth Campimeter is 24″ square. It is placed on a table with the patient seated in front, his eye positioned 10½″ from the central fixation point. The operator, standing behind the screen, facing the patient and looking directly into the patient's eye, is able to detect the slightest wavering. The test object is introduced to the visual field from a "Concealment Circle" and is controlled by a magnet from the back of the campimeter. The patient is not able to anticipate or even guess on which radius or from which side, right or left, the test object will appear.

This assures accuracy and eliminates time-consuming rechecking.

The back of the campimeter is clearly marked in degrees of the circle and angles of vision to conform to the standard Berens chart. Each radius tested is immediately entered on the chart . . . No markers to be placed or removed.

The time saved in each examination creates opportunities for more "fields" or other office work.

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